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THE
PRINCIPLES AND PRACTICE
OF SURGERY

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THE PRINCIPLES AND PRACTICE OF SURGERY

BY

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PART VIII

THE SURGERY OF DEFORMITIES

CHAPTER I

GENERAL CONSIDERATIONS

Deformities may be *classified* as: Those developing *before birth* (congenital); those resulting from injuries *during birth*; those developing *after birth* from various *causes*.

Congenital Deformities.— *Congenital deformities* may be divided into three groups: Those developing from *atypical development of the embryonic anlage;* those due to *abnormal positions of the child;* those due to *diseases developed during intra-uterine life.*

Deformities Due to Atypical Development.— The group of deformities due to atypical development of the embryonic anlage may be exemplified by the occurrence of *six or more toes* and by a certain form of *scoliosis* due to the presence of an adventitious vertebra. There would seem to be little doubt that the causation in this group of cases is attributable to unknown hereditary influences.

Deformities Due to Faulty Position of the Fetus.— Deformities due to faulty position of the fetus form the largest number of the various kinds. Their *causation* is attributable to *contracted pelves* and to *lack of amniotic fluid.* This faulty development is easier to understand in cases in which the child exhibits *multiple deformities;* with *single deformities,* the explanation is not so clear. In the latter instances, *strangulation by amniotic bands* is adduced as the causative factor. To this group belong club foot, congenital dislocation of the hip, etc.

Deformities Due to Intra-Uterine Diseases.— Of the diseases developing during intra-uterine life which are responsible for deformities, those of the skeleton, especially chondrodystrophia and osteogenesis imperfecta, are the most important.

Deformities Developed as a Result of Injury During Delivery.— This group may be exemplified by *wry neck* which follows rupture of the sternomastoid muscle; by *cerebral hemorrhage* and its sequential *spasticity of the extremities* (Little's disease); and by injury to the *brachial plexus* and consequent paralysis of the arm, etc.

Deformities Acquired After Birth.— *Rachitis* is by far *the most frequent cause of acquired deformities.* This disease lessens the resistance of the bony structure and is responsible for *flat foot, bow legs, knock knees, coxa vara, contracted pelvis, scoliosis,* and *kyphosis. Osteomalacia,* the disease which v. Recklinghausen has shown to be *identical with rachitis* (Part V, chap. viii), is responsible for similar deformities occurring in the adult, especially in women. The *other causes* relate to *inflammatory processes of bones and joints* (osteomyelitis and arthritis) (Bonnet,[1] Koenig,[2] Ludloff[3]), and to *paralyses,* which include those of infants and the group of paralytic joint contractures (Seeligmüller[4]). In this category belong also *deformities due to apparel,* such as *hallux valgus* (from shoes); *X-legs* (from garters); *wasp waist* (from corsets), and *talipes equinus* (from bedclothes). So-called *overload deformities,* such as flat foot, scoliosis — with and without kyphos — are regarded by many observers (see Lange[5] and his bibliography) as due to *late forms of rachitis.* On the other hand, Lange[5] seems to have shown that "muscle pull" will cause certain deformities of the foot (hollow foot), and that coxa vara and scoliosis may be due to postural causes (policemen, waiters, etc.). Lange[5] believes that in these cases circulatory disturbances are responsible for lessening of the resistance of the affected bones, rendering them liable to deformation — a view which is supported by the histological findings of Julius Wolff.[6] In the work of the latter, the views of v. Volkmann[7] and Hütter[8] are convincingly controverted in this connection. *Statistical data* regarding acquired deformities collected by Biesalski are presented by Lange.[5]

The *diagnosis* of fully developed deformities is usually easy. *In the early stages* of their development great care and systematic examinations are essential to a correct conclusion.

Inspection includes comparison with opposite normal parts, the observation of the condition of the skin, observation of the presence of *swelling, and atrophy of the soft parts.*

Palpation is directed toward discovery of differences in temperature and the recognition of palpable masses, areas of thickening and cavities.

Measurements are particularly valuable. While these are taken up in connection with special injuries (Fractures, Part IV, chap. iv) and deformities, allusion may be made at this time to the Roser[9]-Nélaton[10] line, as indicating the importance of their employment.

The *posture assumed by joints and their range of motion are determined* by the use of special instruments or, which is very satisfac-

tory, by *tracings* on paper of the outline corresponding to the affected part.

The *Röntgenogram* is of incalculable value in the diagnosis of deformities. However, it must always *verify the clinical diagnosis — never indicate it.*

BLOODLESS OPERATIONS.— So-called bloodless operative measures of relief, such as *redressment of arthrogenous contractures, infraction, epiphyseolysis, and reposition of congenital deformities,* are frequently employed in orthopedic surgery.

Redressment of arthrogenous contractures may be exemplified by the bloodless correction of flexion contracture at the knee joint and adduction contracture at the hip joint. In children the hands of the surgeon may be used for this purpose; in adults, however, the use of apparatus is necessary. Various more or less satisfactory forms of apparatus are used by different surgeons and for different purposes. In recent years the tendency has been to use especially constructed tables, of which the one of Gaugerle, shown in connection with the mechanical treatment of kyphosis (Fig. 719), may be regarded as a type. Apparatus of this sort possesses the advantage of making it feasible to standardize accurately the degree of correctional force employed. The work of Lange[5] depicts several of these tables, among which his own is not without merit.

Redressment of an arthrogenous contracture *consists in stretching the parts on the concave side of the joint,* i. e., the capsule, the tendons, and the skin. The *procedure is not without danger,* especially of *injury to the vessels and the nerves.* These are most likely to be traumatized in redressment of the knee and ankle joints. The vessels are less vulnerable than the nerves. SUFFICIENT FORCE TO CAUSE CYANOSIS OF THE PART IS TOO GREAT. UNDER NO CIRCUMSTANCES SHOULD A CYANOTIC LIMB BE IMMOBILIZED; under these conditions efforts at redressment must be abandoned and the open operation resorted to. Another danger of redressment in connection with club foot is worthy of mention. The stretching force may tear the subcutaneous connective tissue and the resultant effusion of blood may become infected through an invisible rupture in the epidermis, especially when the latter is already in a pathological state. For this reason the skin of the part and the hands of the surgeon should be cleansed as for operation (Part I, chap. xi). Aside from the local menace involved in redressment, certain general dangers must be taken into account.

The *general dangers of redressment* of contractures are evinced by

disturbances of consciousness and convulsions, sometimes resulting in death. Since the work of Payr,[11] these manifestations have been generally ascribed to *fat embolism* (Part IV, chap. xiii). However, the occurrence of convulsions is ascribed by Codevilla[12] and others to overstretching of the nerves and to the psychic influences of the manipulations. Biesalski[13] has observed glucosuria, fever and somnolence without convulsions.

In any event, the surgeon must be reconciled to carrying out a prolonged after treatment, which means the employment of gypsum or other apparatus, perhaps for years. This, together with the dangers stated and the tendency to recurrence of the deformity in a not inconsiderable number of cases, has caused the trend of recent years to incline toward the use of the open operative methods of relief.

Infraction, in modern orthopedics, is used *mostly* for the purpose of *correcting rachitic deformities.* The purpose may be accomplished with the hands, the bone being bent over the edge of a table. However, the infraction is best produced by means of suitable apparatus. Many forms of apparatus have been devised for the purpose, of which the osteoclast shown in Fig. 634 may be regarded as a type. In orthopedic institutions an especially constructed table is generally used.

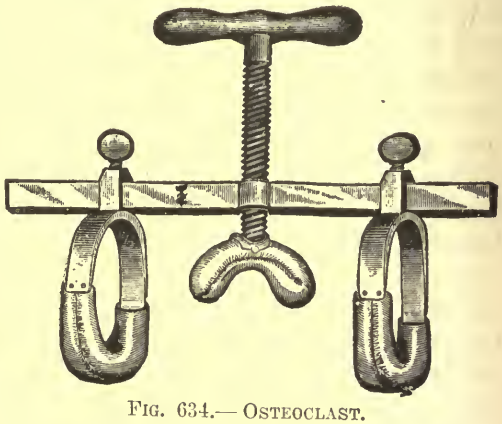

FIG. 634.— OSTEOCLAST.

Theoretically, the dangers of infraction should be the same as those discussed in connection with redressment (see above) and, clinically, this is true. However, strange to say, the complication so greatly feared in redressment — fat embolism — rarely occurs in infraction (though it should be more frequent), and, as a matter of fact, the latter is rarely followed by untoward developments.

Epiphyseolysis consists in a bloodless epiphyseal separation. It is rarely performed in this country, though Italian surgeons employ it for the correction of genu valgum. Its technic is taken up with the discussion of genu valgum (p. 1390).

Reposition is chiefly employed for correction of congenital disloca-

FIG. 635.—HOLLOW GYPSUM BANDAGE MODEL OF THE UPPER ARM.

tion of the hip. It is extensively discussed in the chapter dealing with this subject.

The general considerations regarding *bloody operations of orthopedic surgery* do not differ from those discussed in connection with operations in general, such as asepsis, control of bleeding, etc., nor from those of general operative procedures (such as tendoplasty, osteotomy, arthrectomy, etc.) described in the verious parts of this work.

The *technic of orthopedic dressings* comprises the use of the same materials used in connection with the treatment of fractures (Part IV, chap. iv-v) and those spoken of in connection with spondylitis. The fact that in the class of cases under discussion, dressings and apparatus must remain *in situ* for a long time calls for the use of considerable skill in their adjustment and care in the selection of material suitable to the demands of the individual problem.

For extension, *adhesive plaster* that causes the least irritation to the skin should be employed. This, in a measure, is determined by the formula of the adhesive mixture with which the plaster is coated; one containing zinc is regarded as the least irritating. Before the plaster is applied the skin should be cleansed and hairy surfaces shaved.

The *starch bandage* may be used for the immobilization of the extremities of children.

FIG. 636.—POSITIVE GYPSUM MODEL OF THE UPPER ARM.

The *gypsum dressing* is the one most frequently selected. Unfortunately, serviceable gypsum bandages are not readily obtained in the market and their preparation by unskilled hands is not satisfactory. *Crinoline*, in which finely powdered gypsum is incorporated *by hand*, gives the best results. Before application the bandage *should be immersed* for one minute in a *solution containing 25gm. of powdered alum to a liter of water.*

FIG. 637.—FELT REMOVABLE SPLINT MOULDED OVER POSITIVE GYPSUM MODEL OF UPPER ARM.

The posture of the patient should permit the uninterrupted application of the gypsum dressing and its subsequent hardening without change of position. Until the latter is accomplished, the patient should not be transported.

FIG. 638.—HESSING'S PELVIC AND LEG APPARATUS.

Liquid glass (sodium silicate) *reinforcement* of the gypsum dressing consists in painting the solution of silicate on the gypsum one or two days after the latter is applied. The result is a hard, firm and very satisfactory dressing.

The *removal of the gypsum dressing* is a problem of some importance. As a rule, this is done by the junior assistant on the house staff, who is usually armed with a pocket knife, backed by youth and fear of the "Visiting." Special instruments, including an electrically driven circular saw, have been devised for this purpose. It is desirable that the problem receive proper consideration, especially when removable apparatus is employed. It is also suggested that the use of a proper armamentarium lessens the necessity for frequent renewal of the dressings.

Proper tools are especially necessary in the preparation of a model of the afflicted part. In the *preparation of the gypsum model,* the skin is anointed with vaselin or a similar substance and the gypsum bandage applied in the usual manner, *with the part in the corrected position.* Before the gypsum has quite hardened, the dressing is cut down and removed and the slight deformations, due to this manipulation, are corrected by means of a circular gauze bandage. Fig. 635 shows the condition of affairs (*negative model*) at this stage of the proceedings. The *hollow negative model* is now filled with gypsum paste and the *positive model* (Fig. 636), in accord with which any desired apparatus may be fashioned, is obtained (Fig. 637).

Fig. 639.— Hessing's Scoliosis Corset.

Figs. 635–637 show a very simple aspect of the problem, which may, however, be regarded as illustrating the principle involved in the procedure. Acquaintance with it enables the practitioner to obtain a replica of the afflicted part and send it to the technician for the construction of the necessary apparatus, which may be conveniently replaced at any time.

Orthopedic appliances are constructed of various materials, including steel, aluminum, canvas, leather, etc. The most important factor in their construction is that they should conform as closely as possible to the outline of the part to which they are adjusted. Appliances of this sort are often destined to supplement the action of certain muscles and joints or to take their place (artificial hands and legs). They possess the advantage of being easily applied and taken off. Great ingenuity has been exercised in their construction. Fig. 638 shows a

so-called Hessing[14] apparatus used for the purpose of making locomotion possible in a case of partial paralysis of a lower limb; and Fig. 639 shows a scoliosis corset constructed along the same lines. The publication of Gocht[15] gives much valuable information in this connection.

Not a few orthopedists use the so-called celluloid mull apparatus of Kirch (see Gocht[15]), which is light in weight, conforms closely to the body, and may be constructed by comparatively unskilled hands. For the purpose, three parts of celluloid (obtainable in the market) are dissolved in ten parts of acetone; the solution is stirred at intervals for twenty-four hours until a thick, even mixture is formed. The (positive) gypsum model (Fig. 636) is covered with a thick layer of felt or flannel and this in turn covered with a mull bandage. The celluloid-acetone solution is applied to the latter with a heavy brush and the hands; this is followed by another layer of mull bandage and then a layer of celluloid-acetone solution. From seven to ten layers of this sort are applied in accord with the part to be held in the corrected position. *The mixture of celluloid and acetone is highly inflammable.* As apparatus constructed in this way is not firm enough for use in situations subjected to great strain (ambulatory fixation of a lower extremity), Lange" *reinforces the layers of celluloid-acetone mull with steel wires* which he places between them. Figure 640 shows a celluloid "manchette" reinforced in this way.

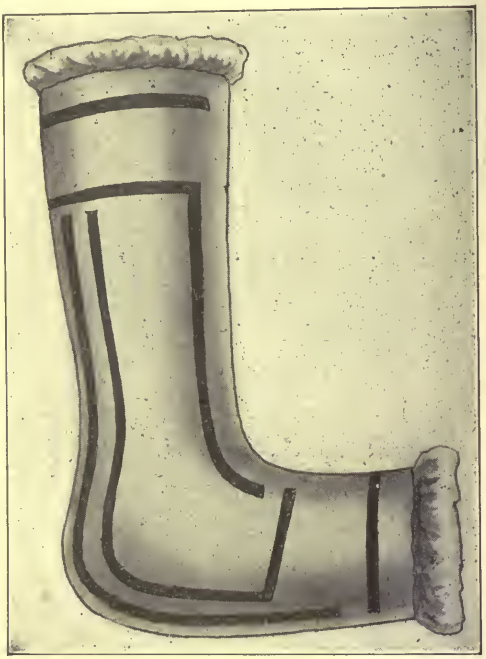

FIG. 640.— CELLULOID-ACETONE SPLINT REINFORCED WITH STEEL WIRES.

Massage, in orthopedics, is destined to increase the flow of arterial

blood to, and the flow of venous blood and lymph from, the afflicted part. It is employed in five ways, (1) *stroking;* (2) *rubbing;* (3) *kneading;* (4) *percussing;* (5) *vibrating.* The first four are performed manually; for the fifth, an especially constructed mechanism is used.

Gymnastics play an important rôle in modern orthopedics. The object is twofold: Either shortened structures are stretched (*passive gymnastics*); or the *musculature* is to be strengthened (*active gymnastics*). In addition to this, frequent motion of joints aims at the *polishing of articular surfaces* (when exudates are present) and at the coördination of movements (in tabes, writer's cramp, etc.). A detailed

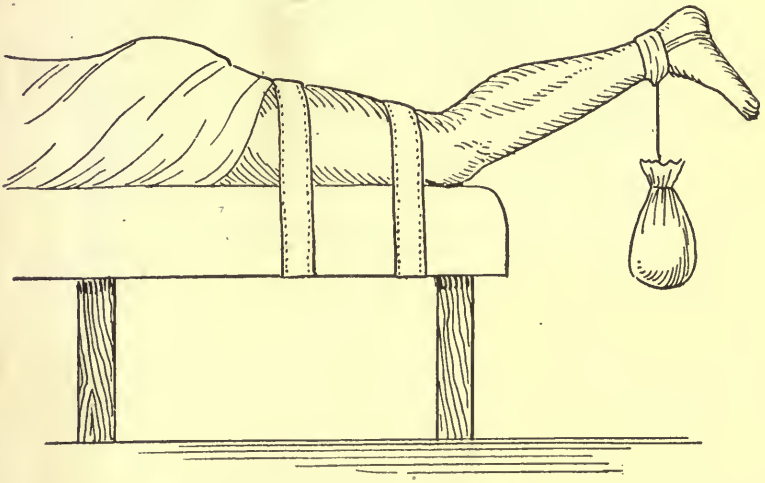

Fig. 641.— Showing the Principle of Passive Gymnastics in Stretching the Knee Joint.

description of the various apparatus used for the purpose is of course impossible here. For this the reader may consult the bibliography at the end of the chapter (numbers 16 to 20 inclusive). However, the principles involved are shown in connection with contracture of the knee joint.

The object of *passive gymnastics* is to stretch the shortened muscles, tendons, and the capsule on the posterior surface of the knee. For the purpose the patient is postured with the abdomen resting upon an upholstered table with the leg extending over the edge. A weight (sand bag) varying from 200 to 2000gm. is attached to the ankle (Fig. 641). *The posture is maintained from five to fifteen minutes*

at least, three times a day. Special apparatus with elastic tension may be used. Figure 642 shows one of these used in connection with the wrist joint.

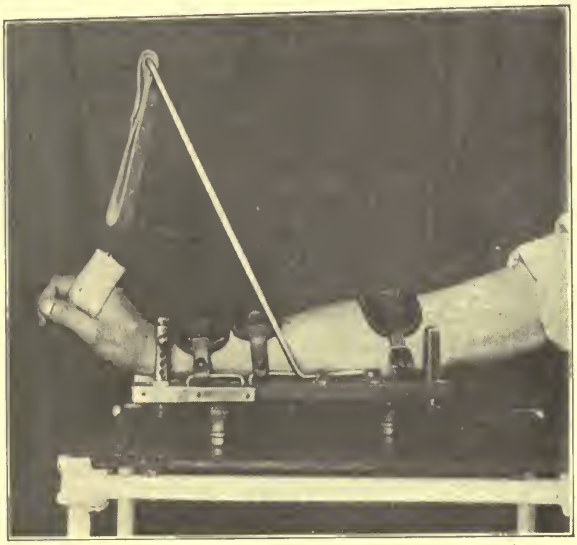

FIG. 642.— APPARATUS FOR STRETCHING THE WRIST JOINT.

Active gymnastics may be accomplished in an equally simple manner. The patient is postured as shown in Figure 643. The weight is

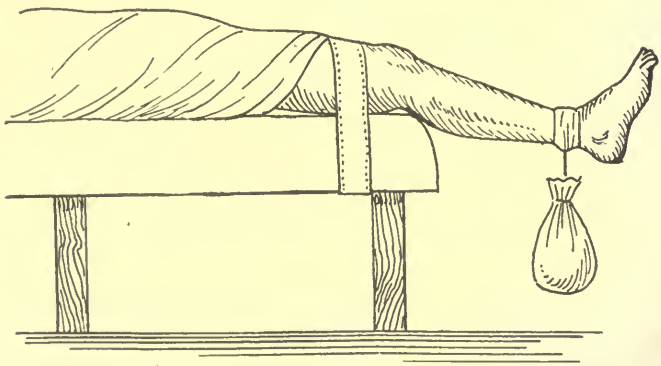

FIG. 643.— SHOWING THE PRINCIPLE IN ACTIVE STRETCHING OF THE KNEE JOINT.

suspended from the ankle and is lifted by the extensor muscles of the thigh. The latter muscles and the tendons and capsule of the anterior

portion of the knee may also be passively stretched with the limb and the weight arranged in this way.

It is of course necessary to arrange the apparatus to conform to the afflicted part. The execution of this does not require much ingenuity when the principle of the method is borne in mind. The need of *especially constructed apparatus* in the treatment of ambulatory cases has led to the development of so-called *mechanotherapy* which, under the impetus given by Zander,[16] has developed into an art of considerable magnitude. This apparatus operates on the pendulum principle and achieves both passive and active gymnastics. Figure 644 shows the apparatus used in connection with the mechanical treatment of the wrist joint. It is shown with the view of illustrating the method. Apparatus for the other parts of the body may be obtained from dealers in surgical supplies.

FIG. 644.— ZANDER WRIST MACHINE.

Heliotherapy occupies the most important place in light therapy. Appreciation of its use in orthopedics is due to the efforts of Bernard[21] and Rollier.[22] While sunlight contains three constituents useful to light therapy (its red and ultrared rays furnish heat; the yellow afford light; and the ultraviolet possess chemical activities), all of its rays are used for therapeutic purposes. The beneficial effect of heliotherapy in diseases of bone is attributed to the mild and prolonged hyperemia it causes. Light treatment is also used in the form of the blue

light of Richter,[23] the quartz lamp, the unilight, and the X-ray. Aside from the beneficial effect of light therapy upon bone tuberculosis, it is also of benefit in the local anemia and lymphatic stasis of poliomyelitic paralyses in children and in rachitis.

Heat in the form of *baths* or *dry heat* owes its virtue to the hyperemia it provokes and is a useful adjunct in orthopedics. For the pur-

Fig. 645.— Tyranauer Dry Heat Apparatus for the Hand and Forearm.

pose of using *dry heat* at a high temperature (baking) the *electric oven* is largely employed. By encasing the part in layers of absorbent cotton, which absorbs the moisture given off by the skin, the temperature may be raised to 300°F. The apparatus shown in Figure 645 is used in treating the hand and forearm. However, apparatus suitable to the application of heat to the other portions of the body is readily obtainable. The active and passive hyperemia of Bier (Part II,

chap. ix) is also used in the class of cases mentioned in connection with heliotherapy.

The therapeutic efficiency of the *X-ray in orthopedics* is limited to its effect upon tuberculosis.

It is probable that the use of the *electric current* is restricted to maintenance of the nutrition of nerves and muscles until anatomical regeneration occurs, and, to a certain extent, to the control of pain.

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CHAPTER II

MALFORMATIONS, DEVELOPMENTAL DEFORMITIES AND SOFTENING OF BONES

As the study of the developmental malformations (*teratology*) is still an indefinite quantity, double and single malformations are characterized in accord with their external manifestations. Excluding the malformations included in the *faulty position of* internal organs, the so-called *monstrosities* and *hermaphroditism, single malformations may be divided* into two groups:

1. Those due to defective development, or *monstra per defectum,* which are manifested either by *incomplete development of the entire body* (*hypoplasia or dwarfism*) or by the absence of, or the incomplete development of, a part or organ (aplasia or agenesis).

2. *Malformations due to excessive development,* or *monstra per excessum,* which are manifested by abnormal size (giantism) or by *increase in the number of various parts.*

The underlying causes of these conditions are already present in the fertilized ovum and resemble the regressive and progressive processes upon which the organic changes of later life are dependent (see also Part VI, Mixed Tumors, etc.).

Malformations Due to Developmental Defects.— These are the result of *mechanical interference with growth,* of *heredity* or of *primary embryonic variations.* These causes determine the occurrence of *aplasia* or *hypoplasia* by interfering with normal development or by arresting it at a certain stage. In addition to this, arrest of development is manifested by a failure of coalescence of embryonic segments and also by parts arising from a single anlage becoming separated and remaining so divided. The latter give rise to *division and strangulation* of parts. Atypical or faulty coalescence may occur as the result of *incomplete differentiation;* and, finally, the arrest of development may be the outcome of lack of space for the fetus and the abnormal position of the extremities during intra-uterine life.

Dwarfism (Microsomia or Nanosomia).— Genuine, or teratological,

dwarfism is a congenital defect of the whole body, originating in the first anlage. It is recognizable at birth and becomes more manifest as life advances. *Congenital dwarfism* must be *differentiated* from the *acquired,* due to diseases such as chondrodystrophia and other congenital systemic diseases of the skeleton — myxedema, cretinism, rachitis, etc.

The *distinctive feature* of genuine dwarfism is its *symmetry,* the pathological variety being characterized by the asymmetrical proportions of the various parts of the body to one another.

The Developmental Defects.— Developmental defects are also *not ascribable to a single causative factor.* They are, of course, not readily proved to be due to mechanical influences. It is easy to understand the *causative influence of bands of adhesions between the amnion and the fetus, which ensnare, strangulate or amputate certain parts.* The latter are called *fetal or spontaneous amputations.* The stumps of strangulated limbs often show rudimentary fingers and toes. The amputated parts are absorbed. *Incomplete* strangulation is characterized by deep incisures in the ensnared part. The *umbilical cord* is capable of ensnaring and strangulating the limbs.

A classification of defects of this sort is offered by Riedinger,[1] as follows:

Amelia — complete absence of the upper and lower extremities. *Abrachius* — absence of the upper extremities. *Apus* — absence of the lower extremities. *Monobrachius* — absence of one upper extremity. *Monopus* — absence of one lower extremity.

Phocomelia (seal limbs) — rudimentary extremities.

Peromelia — severe deformity of the extremities.

Micromelia — small extremities.

Perodactylia — small fingers or toes; often combined with *bony syndactylia.*

Brachydactylia — shortening of the entire hand. *Brachyphalangia* — shortening of all or of a single finger.

Ectrodactylia — absence of a single finger. *Adactylia* — absence of all the fingers.

Congenital Defects of Single Bones — Defect of the Upper Arm.— Isolated absence of the upper arm has not been observed. On the other hand, it is often underdeveloped when the forearm and hand are developed normally and when the entire limb is defective in size. With the help of prosthesis defects of this sort may be made to functionate to an astonishing extent.

Defect of the Radius.— The radius may be absent from one or both forearms; the former is most frequently the case (Antonelli[2]). It is probable that a small portion of radius is always present. This is demonstrable in the Röntgenogram. As a rule, the entire extremity is more or less atrophic. In most instances club hand (*manus vara*) is present.

Efforts at correction made early in life are followed by improvement. In some instances a certain degree of correction occurs spontaneously; in others, the deformation of the hand increases. The latter may be obviated by *redressment* of the curved ulna and by *mechanotherapy*. After redressment *correctional apparatus* of celluloid or leather should be worn for several weeks. *In adult life* the manus vara may also be corrected by *ulnar redressment*, or by *ulnar linear osteotomy*. The latter is preferable.

Fig. 646.— Congenital Partial Absence of Femur.

Bardenheuer[3] splits the distal end of the ulna and implants the carpus into the fork thus formed, so that the medial side of the splinter rests against the radial side of the carpus and *vice versa*. Both lower splinters are nailed to the carpus. The procedure corrects the deformity and is followed by improvement of function. Methods of this sort (osteoplasty) are preferable to resection of the carpus.

Antonelli[2] employs a somewhat similar, though more complicated, method (see bibliography).

The work of Albee[4] suggests the possibility of implanting a *free bone graft* obtained from the tibia in cases of this sort.

Ulnar Defect.— This is not so frequent as the absence of the radius and is usually complicated by defective musculature. The *diagnosis* is made certain by the Röntgenographic negative. Especial methods of *treatment* are not available. However, those applicable to the radius may be employed.

Defective Femur.— As a rule, imperfect development of the long tubular bones of the extremities is attended with malformations of other portions of the body. Very often the corresponding lower leg is hypoplastic or deformed. The *patella is absent* in all cases, and asym-

metry of the pelvic bones, the thorax, and the vertebral column are frequent coëxisting conditions. Complete absence of the femur is rare; as a rule, a sector of bone is present at the upper end of the tibia.

In cases of this sort the art of the orthopedic technician is of great assistance. The prosthesis shown in Figure 647 is simple and very satisfactory. It is shown here in connection with the same case represented in Figure 646. Fortunately the child was not afflicted with an additional malformation.

Fig. 647.— Prosthesis Applied to Case Shown in Figure 646.

The rôle that developmental defects of the femur play in coxa vara is emphasized by Drehmann,[5] who regards the separation of the upper epiphysis, and consequent arrest of its growth, as responsible for a certain number of these cases.

Tibial Defects.— One or both tibias may be absent, the latter case being the more frequent. The patella is very often absent. As a rule, complete or partial absence of the tibia is complicated by grave *contractures of the muscles of the leg* and *deformity of the foot.* Albert and v. Bramann[6] implanted the upper end of the fibula into the lower end of the femur, a measure which greatly increased the stability of the limb. Huntington[7] implanted the upper end of the fibula into the upper stump of a partially defective tibia with a brilliant result. Stone[8] repeated this with an equally satisfactory outcome. The contractures are treated by stretching. In this situation the employment of prosthesis is particularly useful.

Fibular Defects.— These may be single or double. The tibia is shortened and bent with its concavity backward and toward the fibula. The *foot* is forced into the *valgus* or *varus* position; occasionally, the tibia remains straight.

The *treatment* in these cases is similar to that discussed in connection with the tibia, and comprises the employment of redressment, mechanotherapy, massage, gymnastics, plastic lengthening of tendons, osteotomy, and osteoplastic measures of relief.

Forked Hand and Cloven Foot.— Partial or complete absence of a

single (usually the middle) or of several metacarpal or metatarsal bones and their phalanges causes the hand or foot to be divided into two asymmetrical halves.

The condition is frequently associated with syndactylia and may involve one, two, or all of the extremities. It is probably inherited, though some writers regard it, in some instances, as the outcome of strangulation by amniotic bands.

Fission and Reduplication.— Fission defects are due to failure of coalescence of embryonic segments. This is not rare in connection with the spinal column, of which spina bifida is an example (p. 1422).

Reduplication of a part (Supernumerary parts) is due to the division of a single embryonic anlage or to its separation by strangulation. The result is that there is a twofold development, one member of which, however, is rudimentary.

Fig. 648.— Röntgenogram of Forked Hand.

Fusion Deformities.— Fusion occurs when the embryonic anlage of two organs or parts remain connected, though each develops separately. The phenomenon is attributed to hereditary variations and to mechanical interference with development. The latter comes under consideration when other portions of the body present asymmetrical deformities.

Symmelia is a term applied to complete or incomplete fusion or faulty separation of the lower extremities. It is also called "siren formation," a term which, considering its anatomical peculiarity, does not appear particularly appropriate. As a rule, it is attended with more or less arrest of development of the feet.

Synosteosis, or coalescence of the bones of one or both forearms, is occasionally met with. The condition has been studied by Biesalski,[9]

Lieblein,[10] and others. The work of Riedinger[1] may be consulted also to advantage.

When the exact condition of affairs is revealed by the Röntgenogram, it is not infrequently possible to establish a certain degree of function by means of osteotomy, redressment, and osteoplasty.

Syndactylia.— Syndactylia is a congenital fusion of the fingers. It occurs in manifold forms and is often combined with other deformations and with partial giantism (Fig. 649) or contractures.

The mildest form is that of *web finger*, in which the fingers are par-

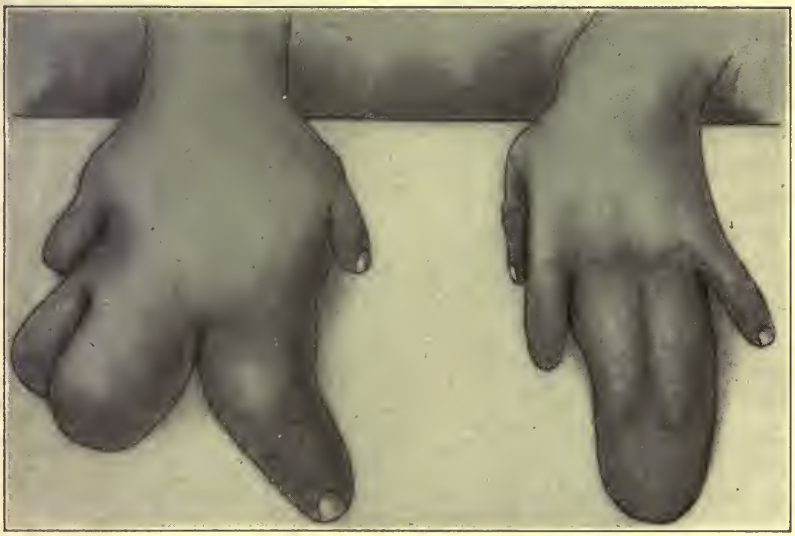

Fig. 649.— Syndactylia and Partial Giantism.

tially or completely connected with one another. In severe cases the bones are united.

Operative separation of the fingers is made difficult by insufficient skin. The principle underlying efforts of this sort is shown in figure 650. Several additional methods of procedure are described by Jacobson and Steward.[11]

Malformations Due to Faulty Posture of the Fetus.— In this category belong the malformations due to insufficient amniotic space, such as *club foot and congenital luxations.* However, most writers classify the former with developmental anomalies.

Malformations Due to Excessive Development.— Malformations of this class are dependent upon intrinsic causes, *i. e.*, an increase in the quan-

tity of anlage material (hypertrophy) or of developmental energy (hyperplasia). When abnormal growth involves the entire body, the term *genuine giantism* is used; when it is restricted to certain areas, the term *partial giantism* is employed. *Leontiasis ossea, congenital elephantiasis of the skin, and macrodactylia* belong to the latter class. In some instances, the process appears in the form of multiple exostoses.

Giantism (Macrosomia).— In *genuine giantism* the entire body is symmetrically enlarged, as a rule, *from the time of birth,* but the condition may *develop later in life,* i. e., about puberty. Cases not ascribable to congenital causes are not properly regarded as genuine instances of giantism (Schwalbe[12]). Most persons afflicted with giantism possess lowered resistance to disease and are easily fatigued. According to Riedinger,[1] this suggests disturbances of internal secretions, which is illustrated by the fact that early castration is followed by abnormal growth of the bones.

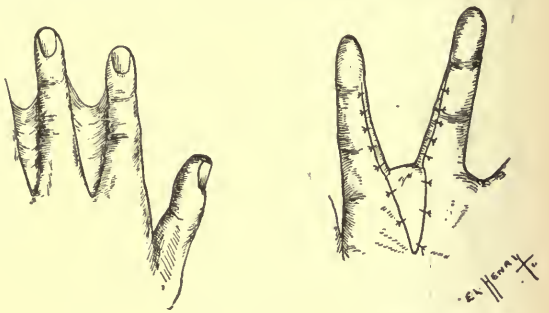

Fig. 650.— Agnew's Operation for Webbed Fingers.

The manifestations of giantism may be limited to one side of the body, or to the lower extremities, or even to a single finger or toe (macrodactylia).

Acromegalia and leontiasis ossea are discussed in connection with hypertrophy of bones (Part V, chap. viii).

Polydactylia.— Hyperdactylia may be single or double. As a rule, the increase is limited to a single phalanx, although at times two additional phalanges are present (Joachimsthal[13]). The adventitious phalanx is often very rudimentary (Fig. 651). It is generally amputated with ease.

Developmental Disturbances of Bones.— Fetal diseases of bones occur as soon as the ovum has reached a certain stage of development. The skeletal diseases of the fetus do not differ from those of postuterine life. They are, however, more likely to be attended with grave malformations when they occur during intra-uterine life.

The Congenital Systemic Diseases of the Skeleton.— To these

belong *chondrodystrophia fetalis, osteogenesis imperfecta, athyreosis congenita* (*congenital myxedema*), *osteochondritis syphilitica,* and the *multiple cartilaginous exostoses* and *enchondromata.*

Chondrodystrophia fetalis is really a *fetal rachitis.* It does not differ materially from that described in connection with diseases of bones (Part V, chap. viii). The fetal form of the disease is extensively discussed by Frangenheim[14] and by Siegert,[15] who furnish complete bibliographies on the subject.

Osteogenesis imperfecta consists in *congenital incomplete ossification*

FIG. 651.— POLYDACTYLISM.
(Symmers, Bellevue Hospital case).

of the bones, or in a meager formation of osteoblasts of the medullary substance and the periosteum. The bones remain *thin* and *soft,* or are *brittle and porous* (*fragilitas osseum* and *osteopsathyrosis congenita*). As a rule, death occurs *in utero* or soon after birth.

Athyreosis congenita (congenital myxedema) represents a disturbance due to a *congenital defect of the thyroid gland.* It is not related to the myxedema of adult life. It is often associated with congenital cretinism. The *clinical picture* is very similar to the latter — apathetic facies, stupid expression, large tongue, hoarse voice, low temperature and dry skin. After a time the skin evinces the characteristics

of a genuine myxedema. The *pathological picture* is that of arrest of bony development.

Mongolism is a condition of *congenital idiocy* in which the features bear some resemblance to those of the Mongolian race. In addition to various malformations (hypoplasia), the joints are lax and the skin is myxedematous. The imbecility is likely to be "joyful" (merry imbecility). Its cause is unknown.

Infantile myxedema begins about the fifth year of life (Siegert[15]) and is due to a genuine *hypothyroidism,* the outcome of an acute infectious disease. It is attended with arrest of skeletal development. The disease is not well understood (see Frangenheim[14]).

Osteochondritis syphilitica, multiple cartilaginous exostoses and enchondromata, atrophy and hypertrophy of bones and softening of bones (rachitis and osteomalacia) are all responsible for a number of deformities. These are taken up in connection with Diseases of Bones (Part V, chap. viii). For detailed discussion, the authors mentioned at the end of the chapter (No. 16 to No. 21, inclusive) may be advantageously consulted.

Of the *chronic inflammatory* processes involving the skeleton, tuberculosis (Part II, chap. xxiii) is responsible for the greatest number of deformities, followed, in the order mentioned, by syphilis and chronic osteomyelitis. The same order is applicable to *diseases of joints* (Part V, chap. viii), especially in connection with *ankylosis* and *arthrogenous contractures.*

In addition to this, deformities are the outcome of chronic inflammatory diseases (of the same class) of muscles, tendons and bursae.

The rôle that *acute inflammatory processes* of bones, joints and the soft parts play in the production of deformities is also taken up in connection with the discussion of diseases affecting those parts. However, these are subjected to the same measures of relief addressed to the deformities due to chronic processes and, therefore, will not be given special consideration at this time.

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CHAPTER III

CONGENITAL LUXATIONS AND CONTRACTURES

Congenital Dislocation of the Hip.— Congenital dislocation of the hip was known to Hippocrates. About 1700 Verduc studied the problem on the cadaver with the view of developing a method of reduction. In 1830 Humbert accomplished reposition on the living. Late in the nineteenth century, Roser and Reyher recommended and practiced excision of the head of the femur, and Paggi deepened the acetabulum after exposing it. Soon after this Hoffa and Lorenz perfected the open operation. To the latter belongs the credit of placing bloodless reposition on a sound basis, as the outcome of what Gaugerle calls "the painful but therapeutic espionage of the Röntgenogram."

FIG. 652.— RÖNTGENOGRAM OF CONGENITAL LUXATION OF HIP.

The *etiology of congenital luxation of the hip* has been made the subject of numerous hypotheses. At present two are regarded as determining *developmental anomalies*, and *mechanical factors*, the latter occurring *in utero* (faulty position of the fetus).

Displacement of the head of the bone is made possible by stretching of the capsule, which is, however, attended with important alteration of the skeleton and soft parts.

The *skeletal alterations* may be studied in connection with the Röntgenogram, especially the stereoscopic negative. However, as in all Röntgenograph negatives, the interpretation is not always a simple matter.

At first, according to Lange,[1] the caput femoris (in most cases) is luxated upward. It may (according to Kölliker and Hoffa[2]) remain

in this position (*typus luxationis supracotayloidea*). Usually, however, under the influence of the weight of the limb and stretching of the capsule, it is converted into a posterior upward luxation (*typus luxationis supracotyloidea et iliaca*). In the last stage the head of the bone rests directly behind the ilium (*typus luxationis iliaca*) (Fig. 652).

FIG. 653.— DOUBLE CONGENITAL LUXATION AT HIPS.

Note marked lordosis and pelvic tilt.

The pelvis, as the result of the femoral luxation, is forced into an abnormal position (*compensatory pelvic tilt*). This is especially noticeable in double luxation (Fig. 653). In addition to this, the *pelvic bones* and the *femur undergo atrophic changes.*

The *head of the luxated femur* shows a distinct *hypoplasia,* the neck is affected in a similar manner, and the shaft also atrophies, but to a less degree. The hypoplasia of the caput progresses year after year and by the time adult life is reached, it may be quite flattened out, resembling a "buffer" on a railroad car (Fig. 654).

In many cases the neck of the bone undergoes deformation. This takes a variety of forms, such as *bending, twisting, flattening, shortening,* etc.

The *changes in the pelvis* are usually congenital and consist in hypoplasia of the upper portion of the acetabulum. The roof of the latter is not broad and excavated, but presents a steep obliquity, which very often produces an indenture into the caput (the "glide furrow" of Ludloff[8]). The *floor of the pan,* on the other hand, is hypertrophied and is broader than that of the normal side. This condition of affairs produces a flattening of the acetabulum. The acetabulum is never absent (Hoffa[4]). As life progresses the changes increase, until ultimately little of the outline of the acetabulum is discernible. The anatomical peculiarities in this connection are clearly depicted in a series of illustrations in the work of

Gaugerle.[5] At the situation where the luxated caput is fixed an adventitious acetabulum is formed (Fig. 655) (see also Ludloff[3]).

The *changes in the soft parts* consist, of necessity, in *shortening of the capsule,* and the *muscles,* in accord with their location, are either *shortened or lengthened.* In either event, they ultimately become atrophic. The condition of the soft parts greatly interferes with reposition.

The *clinical picture of unilateral luxation* at the hip *varies somewhat from that of the bilateral lesion.*

A B

FIG. 654 *A* AND *B*.— BUFFER DEFORMITIES OF HEADS OF FEMURS AFTER PROLONGED LUXATION (CONGENITAL).

The smaller the child, the less manifest are the typical symptoms. In nursing infants they frequently escape notice, but become more evident as the child begins to walk and the body sinks toward the affected side. LOCOMOTION IS CHARACTERISTIC and is NOT AN ORDINARY LIMPING GAIT, BUT MAY BE CHARACTERIZED AS "WADDLING" caused by the shortening of the limb and the movements of the luxated femur.

The shortening varies from one quarter to one half centimeter at first, gradually increasing to from four to eight centimeters in a few years. As this increases, the child attempts to compensate for it by tilting the pelvis and walking on its toes. An important symptom is the *Trendelenburg phenomenon, i. e.,* when the child stands on the affected side, the sound buttock sinks down (Fig. 656A), and when it stands on the sound side, the affected limb is held rigidly perpendicular (Fig. 656B).

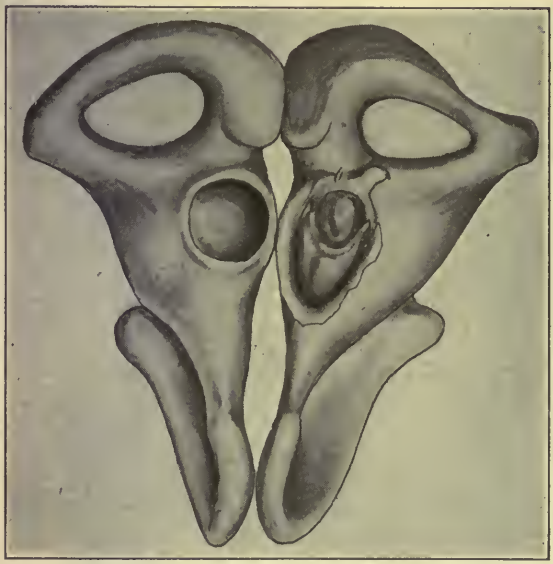

FIG. 655.— On the Left, ADVENTITIOUS ACETABULUM, ELONGATED INTO A TRIANGLE; on the Right, A NORMAL PAN.

As the outcome of the shortening, a *static scoliosis,* with the convexity toward the affected side is developed.

In the posterior type of luxation the normal resistance imparted to the fingers by the head of the bone behind the femoral artery is absent and a distinct hollow is present. In the anterior type the caput is more prominent than is normal. For the purpose of determining the location of the trochanter, the *Roser-Nélaton line* (p. 625) is of service. The elevation of the trochanter corresponds, in a general way, to the degree of shortening of the limb. In the tests care must be taken to maintain the pelvis on its normal level. The *abnormal motility of the head of the femur* and the increased rotary excursion of the trochanter are of diagnostic importance.

The *symptoms in bilateral luxations* are the same as those of the unilateral — much exaggerated. The dominant symptom is *lordosis* in the lumbar region. The latter is caused by the marked anterior tilt of the pelvis, which is secondary to the malposition of the heads of the femurs. The lordosis disappears when the patient is seated. Locomotion is distinctly "waddling," not limping.

The *prognosis*, without treatment, is unfavorable. When an adventitious acetabulum is formed near the normal site of the pan, locomotion, with the aid of an artfully constructed prosthesis, may be satisfactory. Even in these cases more or less pain persists during life.

The *treatment* is divided by Lorenz[6] into *radical* and *palliative*. The former aims at complete restitution to the normal; the latter is

A B

FIG. 656.— TRENDELENBURG PHENOMENON IN CONGENITAL LUXATION OF HIP. On the left the gluteal musculature is unable to hold the pelvis level.

directed toward elimination of pain, contractural deformity, and limping.

THE RADICAL TREATMENT.— Bloodless reposition by the *Lorenz method* is carried out by the *lever maneuver*. Reposition is accomplished over the posterior rim of the acetubulum.

The patient lies supine on a table with the legs hanging over its edge. An assistant fixes the pelvis with the sound limb in maximum flexion at the hip. *The luxated limb is flexed sagittally, at the hip*

joint 90°, in the knee 30°. In this way the shortened adductor, the muscles of the tuberosities and the iliopsoas are relaxed, and a *sudden, sharp pull, in the direction of the long axis of the flexed thigh, brings the caput femoris close to the posterior rim of the acetabulum (first phase of reduction — flexion 90°).*

Next, the shortened muscles are stretched by *carrying the flexed thigh outward to 90° of abduction,* so that it lies parallel with the top of the table (*second phase of the reposition — abduction 90°*).

With the hand of the operator, or a fist, or a wedge, placed beneath the trochanter to act as a fulcrum, the other hand makes firm gradually increasing pressure on the knee (toward the floor), thus forcing the head of the bone against the isthmus of the capsule, widening it, and at the same time lifting the head of the bone over the edge of the acetabulum.

Hoffa[7] and Schanz[8] use the pendulum method. Lange[1] uses an extension method, employing a special table not unlike that of Hawley, and a lever for the purpose (see bibliography).

Retention of the reposition is the most important part of the blood-

Fig. 657.— Fourth Phase of Reposition Maneuver in Congenital Luxation of the Hip (Lorenz Method) (Whitman).

less method of Lorenz, whose work in this connection is particularly praiseworthy.

In the primary dressing the thigh is maintained in 90° abduction and 90° flexion. The knee is flexed at right angles to the thigh (Fig. 658).

The *application of the gypsum* must be carefully carried out. The patient is placed on a pelvic support; the bony prominences are lightly padded and the gypsum applied evenly and thickly. This is best extended over the areas shown in figure 658. At the end of five or six weeks the area of the apparatus may be reduced, as is shown by the black lines in figure 658. Attention to detail in the care of the

child, especially with regard to cleanliness, is important. The posture in bed should obviate deformation of the apparatus. At the end of from two to five months the primary dressing is removed and a *secondary apparatus* is applied. If at this time the head of the bone has remained reduced and the joint evinces some rigidity, *abduction is*

FIG. 658.— FIXATION IN THE LORENZ PRIMARY POSITION.

For greater security the knee on the affected side and the sound thigh are included in the gypsum. The dotted lines show the limits of the dressing employed by a number of orthopedists.

lessened 40° and the limb is rotated inward. The secondary apparatus conforms to that outlined by the black lines in figure 658. It is true that not a few more or less ingenious apparatuses have been constructed to replace the gypsum for the secondary dressing. However, ordinarily the latter is the most readily handled. Secondary immobilization is maintained for ten weeks or more.

The *treatment following removal of the gypsum dressing is* based on what is accomplished during the period of retention. Lorenz[6] had hoped that locomotion in the corrected position would cause the head of the femur to deepen the acetabulum. Springer and Weber[9] have shown that during this period no considerable reformation of the acetabulum takes place, and the latter does not show marked alteration in this connection for from one to two years after the reposition. The edge of the acetabulum does not deepen while the limb is held still. The *gypsum treatment is simply attended by shrinkage of the capsule (particularly the cap) and of the soft parts,* which prevents reluxation.

For these reasons, the after care is very important. Sudden extension of the leg is to be avoided. Lorenz[6] emphasizes the need of per-

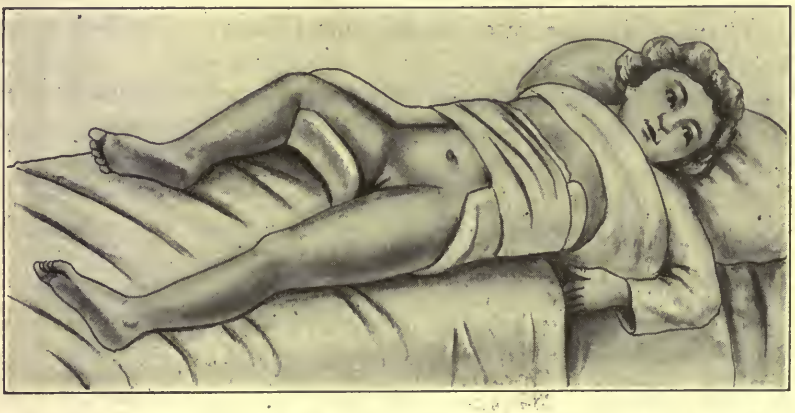

FIG. 659.— GYPSUM "NIGHT SHELL" USED IN THE AFTER TREATMENT OF CONGENITAL LUXATION OF THE HIP.

mitting spontaneous correction of the position of the limb; only in older children is carefully executed redressment and the use of passive correctional motion permissible. In infants the use of a gypsum "night shell," as shown in Figure 659, is advisable. Later, the use of suitable restraining apparatus is a wise measure.

Bloody reposition is carried out along the lines indicated in connection with arthroplasty in this situation (see p. 838; also bibliography).

The *palliative treatment* consists in the application of apparatus which tends to prevent increase of the deformity. These are not particularly successful, but are likely to lessen pain and make locomotion more certain.

Congenital Luxation of the Knee Joint.— This is a rare condition due

to mechanical causes *in utero*. As a rule, only a subluxation is present. The *anterior is the most frequent*. The luxation is attended with grave interference with function.

In young children *reposition by manipulation* (see Dislocation, Part IV, chap. iii) is readily accomplished. Later in life, division of tendons, forcible correction and arthroplasty (p. 838) become necessary.

Congenital Luxations of the Shoulder, the Elbow and the Hand.— These are very rare. None of them presents problems other than those discussed in connection with Dislocations generally (Part IV, chap. iii) and Operations on Joints (chap. vi).

Congenital Contractures.— Congenital contractures per se, that is, those not dependent upon malformations of bones and joints, are very rare. In most of the reported cases, the authors regard the causation as attributable to errors in embryonic development. The *fingers* are most often affected.

The treatment includes gradual stretching, mechanotherapy, and, in severe cases, tenotomy.

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CHAPTER IV

TORTICOLLIS

This deformity (perhaps better called *caput obstipum*) consists in stiffness of the neck and malposition of the head. It is not only *caused by permanent changes* in the soft parts and bones, but also by *temporary unilateral muscle spasm* (v. Alberle[1]). When shortening of the sternomastoid muscle causes the malposition, the head of the patient is inclined toward the affected side and the face is turned toward the healthy shoulder. In the other classes of cases the abnormal position is not as typical and varies with the underlying pathological causation, so that bending, twisting, elevation and depression of the head may be presented. In addition to this, a certain number of cases are ascribable to nervous influences and are called *functional or nervous torticollis,* in contradistinction to the others, which are designated as *organic torticollis or caput obstipum.*

The lesion may be divided into three kinds, *torticollis developed before birth, during birth, and after birth* (v. Alberle[1]).

Of those developed before and during birth, the former are the result of *lack of room in the uterus,* which causes (1) the head to be fixed in an abnormal position and subsequent contracture of the sternomastoid muscle; (2) faulty development in the *embryonic anlage;* or (3) *congenital osseous changes in the cervical vertebrae.* The last consists in (a) coalescence of the atlas with the occipital bone; (b) synostosis of the atlas with the epistropheus; (c) coalescence of several cervical vertebrae; (d) the presence of a cervical rib; (e) the presence of adventitious vertebrae in the cervical or dorsal regions. V. Alberle[1-2] depicts some startling specimens of this class of cases (see bibliography).

Those caused during birth are the result of *trauma during delivery,* especially during difficult births (breech presentation), in which the sternomastoid muscle is subjected to undue strain. The injury consists in either *stretching or partial or complete rupture of the muscle.* In the newly born the site of trauma is characterized by swelling and

pain involving the injured muscle. In many cases, spontaneous healing without deformation occurs; in others, more or less clearly defined torticollis follows. In the latter instance, the process takes on the form of a *chronic myositis fibrosa* (Pincus,[3] Kader[4]).

Of the symptoms, that of *contracture of the sternomastoid muscle on the affected side* is the most striking. The muscle stands out in a hard, short cord, especially when an effort is made to correct the deformity. In this way the head is inclined toward the side of the contracted muscle and turned toward the opposite shoulder; the ear is rotated forward; the chin is somewhat lowered (Fig. 660). In severe cases, the head *in toto* is pushed toward the sound side. *Voluntary motion* of the head is considerably restricted. Viewed from behind, patients with torticollis present the picture of scoliosis.

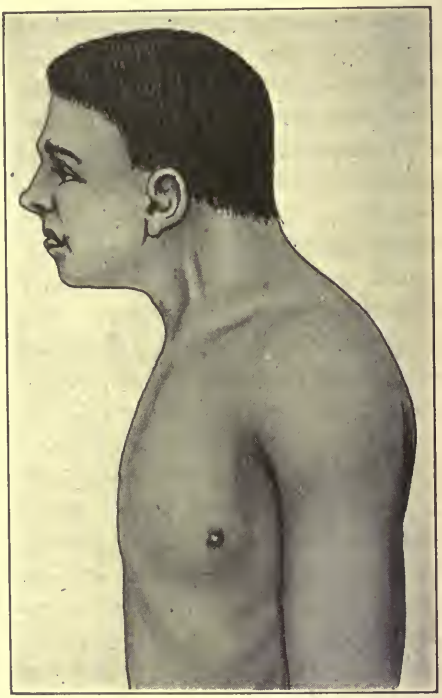

Fig. 660.—Left Sided Torticollis.

The clavicular portion of the sternomastoid muscle is strongly contracted.

The secondary change consists in:

(1) Development of dorsal and lumbar scoliosis.

(2) Asymmetrical development of the cranium and face.

(3) Restriction of the visual field.

The *treatment* during the first six months of life consists in *redressment* into *overcorrection and retention in the latter position.* For the purpose of retention, a *gypsum dressing* (Lorenz[5]), or the *reinforced celluloid wire apparatus* of Lange[6] (p. 1338), may be used. In severe cases, operative methods of relief are necessary.

Open tenotomy is practiced by many surgeons. It is objectionable because of the scar it leaves; on the other hand, it has the advantage of being without danger.

Preliminary tenotomy at the lower end of the sternomastoid muscle, followed by addressment of the vertebral column, is the method of choice.

The tenotomy is performed as follows: The shoulder is elevated and the head is held over the edge of the table by an assistant, thus making the muscle prominent and tense. The tenotome is introduced — flat — between the two portions of the muscle about 1 cm. above its insertion and is advanced to the middle of the episternal notch; the tenotome is then turned toward the muscle fibers and these are divided

FIG. 661.— GYPSUM DRESSING FOR MAXIMUM OVERCORRECTION OF TORTICOLLIS (right side).

with a stroking motion. Division of the fibers is attended with a decided jar. The tenotome is then turned toward the clavicular portion of the muscle and these fibers sectioned in a similar manner. The small stab wound is sealed with collodion. The object of the maneuver is to section the anterior fibers of the muscle while the posterior are torn by the traction made on the head.

The tenotomy is followed by gradual *modeling redressment* of the cervical vertebrae until the scoliosis in this situation is *overcorrected*. The correction is established when the ear on the sound side can be

brought in contact with the shoulder of the same side. In severe cases it may be necessary to expose the muscle and lengthen it by *Z-shaped myoplasty.*

Retention of the overcorrected position is accomplished by the application of a padded gypsum dressing, such as is shown in figure 661, in which the patient remains encased for about ten weeks (Lorenz[5]).

The *after treatment* consists in correctional mechanotherapy, massage, etc. Lorenz swings his patients in a sling (Fig. 716) three times a day and has them wear a "diadem" (Fig. 662) at night for months.

FIG. 662.—"DIADEM" FOR THE AFTER TREATMENT FOLLOWING CORRECTION OF TORTICOLLIS (left side).

Torticollis Developing After Birth.—This is comparatively rare and is classified according to its cause.

1. *Cicatricial torticollis* follows deep burns and severe injuries with loss of substance. It must be corrected by *plastic operative methods of relief.*

2. *Habitual torticollis* is usually *occupational* in origin, but may be due to *errors of vision.* The former is *corrected* by gymnastics; the latter, by the ophthalmologist.

3. *Rheumatic torticollis* is a transient affair and soon disappears under medication and the use of *dry heat.*

4. *Torticollis after infectious diseases* follows scarlatina, measles, and diphtheria, and occurs in the course of lues. It is really a myositis and usually disappears spontaneously.

5. *Torticollis occurs in connection with diseases of the vertebral column (spondylitis), in spastic paralysis and in cerebral paralysis.*

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CHAPTER V

KYPHOTIC ANOMALIES OF POSTURE

This subject opens a vista of far greater range than can be visualized within the horizon of this book. However, it is hoped that the following will indicate to the student the possibilities in this connection and induce him, as he progresses in his work, to give due attention to this subject (see bibliography).

Kyphosis.— Kyphosis may be divided on an etiological basis:

Congenital Kyphosis.— Congenital kyphosis is very likely to be located in the upper segments of the spine. It is the result of embryonic anomalies and of faulty position of the fetus; the latter may be due to amniotic bands (Dieulafé et Gillés,[1] Gebhard[2]).

Degenerative Curved Back.— Degenerative curved back has much in common with congenital curvature, is usually located in the upper segments of the spine, and frequently is *associated with underdevelopment of the body and the mind* (speech). The condition is regarded as *hereditary* and even *racial* (Jewish race). Hoffa[3] thinks that it may be due to faulty innervation of the erector muscles of the back.

Rachitic Curved Back.— Rachitic curved back is explained as follows: When a child less than one year of age, with a feeble erector trunci, is allowed to sit up, it develops a kyphotic deformity at the junction of the thoracic and lumbar vertebrae (Fig. 663). If the child is rachitic, the kyphos becomes a very distinct one. As a rule, both of these conditions disappear spontaneously. However, in not a few instances, the deformity (more or less marked) is permanent. The earlier the child sits up, the lower down is the kyphos.

Relaxation Round Back.— Relaxation round back occurs at the age of the greatest elongation of the spine (nine to thirteen). Lange[4] calls this the round back of school children. No other causative factor need be taken into account, and, as Spitzy[5] says, "*considering the prevalent method of school training, it is astonishing that the condition is not more common.*" He regards the deformity as directly due to faulty posture of the child, though a feeble constitution is the indirect underlying cause. The first indication of the deformity reveals the existence of the constitutional condition and demands that both be

taken in hand at once. Spitzy[5] presents a striking word picture of the process from its inception to the final, permanent deformity (see bibliography). This condition of affairs can be found by any general practitioner who will make the study of the welfare of his young patients the object of proper observation. The avoidance of this condition will save much recrimination (child to mother), many tears, and great unhappiness.

Occupational Round Back.— Occupational round back is determined by the overdevelopment of the flexors of the spine as compared to the erectors. It is usually located in the thoracic vertebrae, though the other sectors of the spine may be the seat of the curve.

Fig. 663.— Severe Form of Rachitic "Sit Kyphos."

Apex at dorsolumbar junction.

The Round Back of Old Age.— The round back of old age is a reversion to the muscular feebleness of early childhood, influenced to a certain extent by the ankylosing effect of chronic arthritis.

The *diagnosis* of round back does not present any difficulty, except, perhaps, in children in whom *differentiation between rachitic kyphosis and spondylitis must be made. The former is influenced by posture, the latter is not.*

The *prognosis* is favorable in all but degenerative and the fixed rachitis forms of the deformity.

The *treatment* relates primarily to *prophylaxis.* This is essentially the duty of the general practitioner, who should instruct the parents to see that assumption of the *erect posture is discouraged, that crawling is encouraged,* and that *breathing exercises and gymnastics in the open air are diligently and intelligently executed.* It is important that exhausting procedures (especially the cold bath, an idiotic practice of modern times) be avoided. The attire of the child should receive careful attention and it should be encouraged to lie on its abdomen. The beginning of school life should be postponed and under no circumstances should a child with a " weak back " be

forced to sit at the piano or at "lap work" for protracted periods of time. Massage of the back is of great service. Later in life the use of gymnastics is perhaps the most helpful measure of relief.

In the employment of gymnastics the character of the exercises suitable to the given problem must be carefully selected. This is a subject of considerable magnitude, to which the student will find special works devoted. That of Whitman[6] is valuable.

FIG. 664.— LEFT SIDED LUMBO-DORSAL SCOLIOSIS.

Apex of the bow in the region of the ninth dorsal vertebra. Rigidity from the seventh to the eleventh dorsal vertebra; overhanging trunk; great torsion; rib hump on the left; concavity on the right side.

Scoliosis — Lateral Curvature of the Spine.— In a general way, scoliosis consists in lateral deviation of the vertebral bodies. However, this is, of necessity, combined with modification of the normal curves of the spine, so that a pure frontal deformation is rarely observed, except, perhaps, in the form of an absence of physiological curves, as in "flat back."

The causation of scoliosis and the mechanism of its development are divided as follows:

CONGENITAL SCOLIOSIS.— Congenital scoliosis is due to asymmetrical development of the bones of the spine and, especially, to the presence of a supernumerary vertebral body, both of which cause the spine to curve. In the latter instance, the convexity of the curve faces the side where the adventitious bone is located (Putti,[7] Garré,[8] Drehmann,[9] Böhm[10]). It is also caused by the influence of skeletal anomalies of parts connected with the vertebrae, such as "high shoulder" (cervical rib), defects of the pelvis, etc. (Garré,[8] Drehmann[9]). Naturally, congenital scoliosis is attended with additional deformations of the trunk and pelvis, the outcome of compensatory postural efforts, and, ultimately, the deformity of the spine becomes fixed and irremediable.

RACHITIC SCOLIOSIS.—Rachitic scoliosis (Fig. 664) probably represents most of the severe forms of this class of deformities. It begins at about the time the child assumes the erect position and influences the entire conformation of its body. As already stated, postural kyphos is often associated with lateral curvature. The apex of the rachitic lateral curve usually corresponds to the dorsolumbar region. THE LATER IN LIFE THE LATERAL CURVATURE DEVELOPS, THE MORE LIKELY IS ITS APEX TO CORRESPOND TO A VERTEBRA AT THE CENTER OF THE DORSAL REGION. In childhood the soft bodies of the vertebrae readily conform to the weight of the erect body. The changes in the vertebrae coincident with rachitic scoliosis are the same as those occurring in the rachitic form of rigid "round back." Rachitis, of itself, merely softens the bones and weakens the ligaments of the spine. Indeed, severe rachitis may occur and the back remain perfectly normal in outline. It must be remembered that, given the presence of rachitis, scoliosis develops as the result of error in the care of the child (Spitzy[5]).

HABITUAL SCOLIOSIS.—Habitual scoliosis corresponds to relaxation "round back," and, as in the latter, its causes are postural.

PARALYTIC SCOLIOSIS.—Paralytic scoliosis occurs in connection with hemiplegia, with weakness of the musculature of the spinal column on one side and overactivity on the other.

STATIC SCOLIOSIS.—Static scoliosis develops in connection with shortening of a lower limb (compensatory scoliosis). *A curvature of this sort may become fixed when it exists for a long time and it is not abolished by the position the patient assumes during rest.*

CICATRICIAL SCOLIOSIS.—Cicatricial scolioses are due to adhesions of scar formation (bands) in connection with suppurative processes within the thorax. To these may be added the so-called *respiratory curvatures* due to irregular action of the diaphragm, in which one side contracts more than the other as the result of pulmonary conditions. This is of course most likely to be the case in rachitic children.

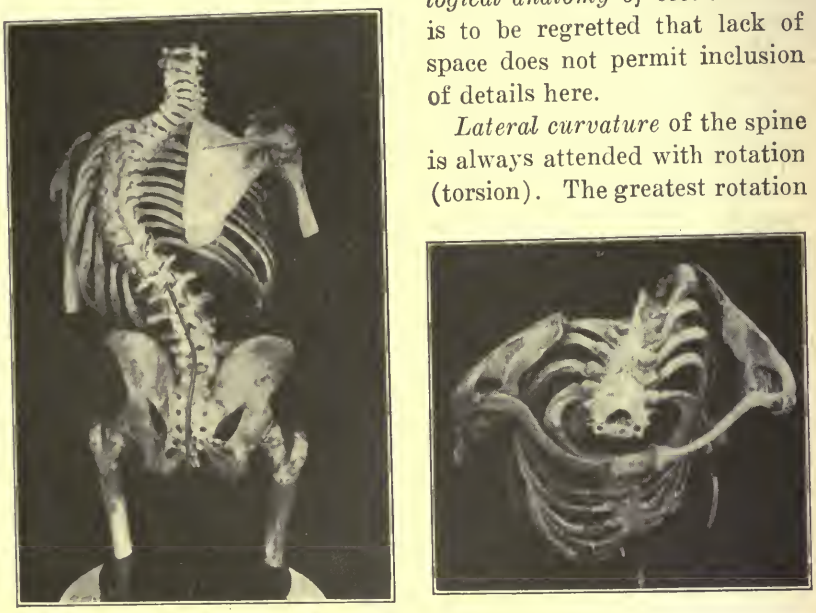
Scoliosis may be due to persistent pain (ischiatic nerve, kidney, lumbago). This is really a postural deformity, the patient assuming an abnormal position in an effort to relieve the pain. Its permanency is dependent upon the persistence of the pain which, in turn, depends upon the underlying cause.

Hysterical scoliosis is associated with lowering of the motor inner-

vation of certain groups of muscles. It is very likely to be transient or intermittent and rarely results in fixation of the deformity.

In all cases of scoliosis the underlying cause must be discovered, if possible. Its removal is not rarely followed by spontaneous disappearance of the deformity (Spitzy[5]).

No problem in surgery has been made the object of greater care and (the word is used advisedly) devotion than that of the *pathological anatomy of scoliosis*. It is to be regretted that lack of space does not permit inclusion of details here.



A B

FIG. 665.— SKELETAL PREPARATION.

A, Severe left sided scoliosis. *B*. The same from above (Lange).

Lateral curvature of the spine is always attended with rotation (torsion). The greatest rotation occurs when the thoracic spine becomes kyphotic. The greater the relaxation, the greater the curve in the spine and the greater the increase in deformity caused by the lever action of the ribs as these become separated from one another. The influence of rotation upon the form of growing vertebral bodies is easy to understand. Their growth and conformation respond to rotation in the same manner as the spiral action of the arm is developed in the human to conform to the erect position. In the upper portions of the bodies of the vertebrae the bony canals are twisted in the direction of the rotation. This

torsion and its attendant changes of necessity affect all the bony parts connected with the vertebral column.

The spinous processes incline toward the side of the convexity. In severe scoliotic deformities a number of combinations of curves occur and the spine winds in and out in lateral deviation, torsion, backward inclination, and kyphotic and lordotic bowing.

The *bodies of the vertebrae* are deformed in accord with the position they occupy in the scoliotic arc, the one at its apex, like the keystone, being wedge shaped, the obliquity of the other bones diminishing as the terminations of the curvature on either end are reached. At the

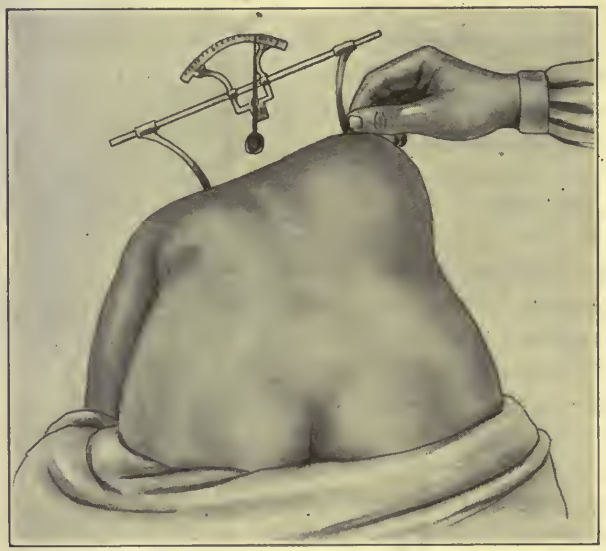


FIG. 666.— THE NIVELLIER TRAPEZE OF SCHULTHESS, TO DETERMINE THE DEGREE OF DIFFERENCE IN THE NIVEAU OF THE THORACIC SKELETON.

outer margin of the wedge the bone tissue is loose and the canals are enlarged (from hydraulic medullary pressure, Nikoladoni[11]). The changes in the vertebrae also *greatly interfere with the motility* of the individual vertebra and the spine as a whole.

The *ribs* undergo a characteristic scoliotic deformity, which is responsible for the most dominant deformation of the trunk as a whole. The torsion of the spine causes the ribs on one side to protrude progressively; on the other (the concave side), they become more or less flattened. This *deformity exercises an unfavorable* influence on *respiration*, an important factor when the affliction is complicated with

lung tuberculosis. The gradual approximation of the ribs, especially in advanced life, causes pressure on the intercostal nerves and gives rise to severe *neuralgia.*

The *pelvis* is influenced because of its relatively rigid connection with the sacrum, which follows the movements of the spine. The diameter of the pelvic cavity is often seriously impaired because of inherent deformations of the pelvic bones and as the result of the encroachment of the distorted spinal column.

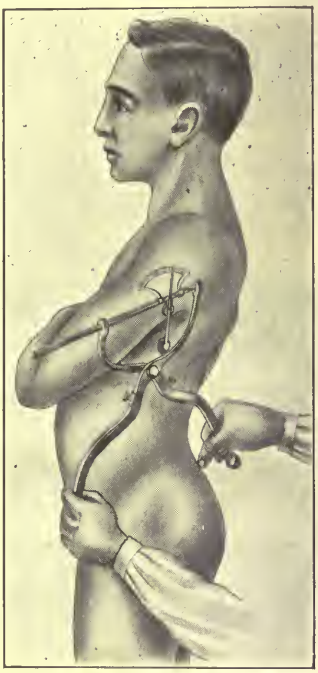


Fig. 667.—Determining the Position of the Pelvis in Scoliotic Deformity by Means of the Nivellier Circle of Schulthess.

The *clinical picture of scoliosis* varies with the form of the curvature. In estimating these deformations, those that are the outcome of faulty posture must be excluded. They are differentiated by the fact that the deformity is not maintained when the patient assumes the position of rest.

All other forms of scoliosis *begin with lateral curvature of the spine* in the "rest posture." From here to *fixation is not a long step. Fixation* is an important determining factor in deciding the presence of scoliosis and is indicated by *modification in the range of the spine.* Movements to the right or left are not accomplished to the normal degree nor are they symmetrically executed. "The alignment of the spinous processes does not follow the motions of the trunk, but shows irregularities in sequence" (Lange[4]). Loss of function is soon followed by *persistence of the deformity when weight bearing is absent.* About this time, symptoms of *torsion* appear and the case soon assumes the type of deformity with which we are, unfortunately, so familiar (Figs. 664 and 665).

The *coincident deformities* consist in elevation of the shoulder and hip. *Locomotion* is attended with limping. The *head* is usually carried to one side.

The *examination and establishment of the diagnosis* are especially

important. Early recognition of the affliction is of inestimable value with regard to treatment and may be considered as determining the prognosis.

The *upper body must be exposed to the trochanters.* The patient is instructed to walk a few steps in order to detect errors in gait. Next, the *locations of the spinous processes* are indicated with a skin pencil. Sudden disappearance of the processes indicates torsion. The *degree of fixation* is determined by observing the outline of the spine in the "rest posture." By drawing the shoulder blades forward, *torsion and deformity of the thorax may be accentuated.* The extent of thoracic deformity may be standardized by means of the *nivellier trapeze* of Schulthess[12] (Fig. 666). The position of the pelvis may be determined accurately by a similar instrument, also devised by Schulthess[12] (Fig. 667).

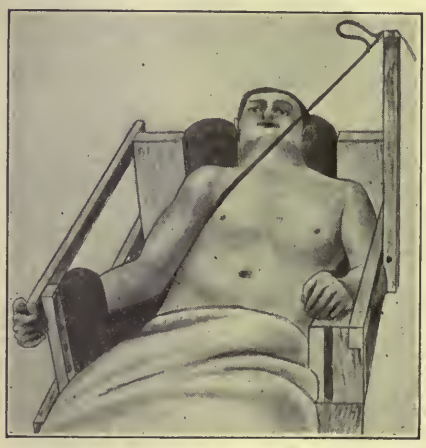


FIG. 668.—LANGE'S CORRECTIONAL POSTURE APPARATUS FOR THE TREATMENT OF SCOLIOSIS.

The traction of the belt overcorrects the deformity.

The *prognosis* in scoliotic processes depends upon their etiology. The congenital forms are the least likely to be improved. Those developed early in life and the paralytic forms also present an unfavorable condition of affairs with regard to ultimate outcome.

Of the *prophylaxis and treatment of scoliosis,* the former has already been discussed in connection with "round back." In this connection the imperative necessity for strict observation of the precautions mentioned is emphasized by the consideration that rachitic scoliosis is attended with changes in the bone which, when once established, are not readily influenced even by the greatest care and skill in treatment.

Passive correction and overcorrection are accomplished in the simplest manner by *suspension* (Fig. 716), which should lift the patient from the floor so that contact with the latter is barely maintained with the toes. This determines the degree of fixation which is not being overcome by the maneuver. The use of the sling with the patient lying

down is less forcible in its effect. This merges into the widely used correctional "posture apparatus," of which that of Lange[13] (Fig. 668) is the simplest. The patient occupies this position for certain periods only of the day. The best results are obtained in cases in which fixation has not yet occurred. The construction of ambulatory apparatus is of course simply a matter of mechanics. One form is shown in figure 669. However, this, like all immobilization apparatus, is objectionable, on the ground that weakened musculature is encouraged in its inactivity. Apparatus of this sort should be used only in connection with correctional gymnastics, massage, mechanotherapy, etc.

Fig. 669.— Redressment Apparatus for Ambulatory Correction of Scoliosis (Lange).

When fixation of the deformed spine has occurred, the problem is amplified. Efforts at its solution have occupied the attention of orthopedists for years. Among the publications devoted to the subject those of Schulthess,[14] Lovett,[15] Spitzy,[16] Roth,[17] Schanz,[18] and Abbott[19] may be studied to advantage.

The work of the last of these (Abbott[19]) has attracted wide attention. Spitzy,[5] whose work in this connection extends over a lifetime, says "The latest step (in correction of scoliotic deformities) has been taken by Abbott, who encases his patients (in gypsum) while in the kyphotic position, on the assumption (which is, according to my experience, a correct one) that this corresponds to the intermediate position of the spine, in which the ligaments are the most elastic and cor-

rection is most likely to be attained.'' It is true that a few orthopedists are disinclined to employ gypsum apparatus in the treatment of scoliosis (Lorenz[20]); others (Schanz,[18] Gaugerle,[21] Whitman[6]) regard it as of great value. Spitzy[5] uses the method only in severe cases, and in these insists that nothing is of greater service than the method as laid down by our own Abbott.[19]

For the purpose, the patient is postured as shown in figure 670. By means of this position and by traction with fabric bands the concave deformity is corrected as far as is possible, and the gypsum is applied as described in connection with spondylitis (p. 1439). A window is cut in the cast over the old protrusion (Fig. 671), and through this are inserted felt pads which, as time progresses, may be increased in thickness. The apparatus is changed every four or six weeks.

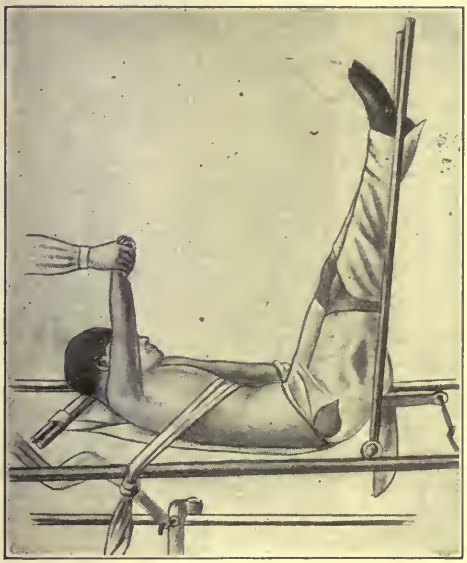


FIG. 670.—POSTURE OF A LEFT SIDED LUMBODORSAL SCOLIOSIS IN MAXIMAL FLEXION AND CONSEQUENT RELAXATION OF THE SPINE (Abbott).

The patient rests on a canvas hammock, which is ''slack'' on the right side so as to permit of protrusion of the body on that side and to attain ''overcorrectional torsion.'' The gypsum is applied in this position.

Of the operative measures of relief in scoliosis, that of Albee,[22] though of recent origin, seems to possess certain possibilities. The technic of the procedure is as follows:

A gypsum bed with firm lateral walls should be molded, before the operation, to the back and sides of the patient's trunk and allowed to harden in the corrected position. Six to eight transverse processes at the apex of the scoliotic curve are exposed by the incision used in spondylitis (p. 1443). The muscles and ligaments over the tips and between the transverse processes are split in approximately equal halves with a scalpel. The transverse processes are split longitudinally into halves, of which the posterior is set over to make room

for the graft. Determination of the graft, the manner of obtaining it, and the means used to place it *in situ* are the same as those described in connection with spondylitis (p. 1443). While the patient is held in the corrected position, the ligaments and muscles are drawn over the site of repair with kangaroo tendon.

The wound is closed with silkworm gut and, after the protective dressing is applied, the patient is bandaged into the previously prepared gypsum bed. At the end of six weeks, a well molded gypsum case is worn for from ten to twelve weeks. After the post-operative period is over, correctional gymnastics, etc., may be employed.

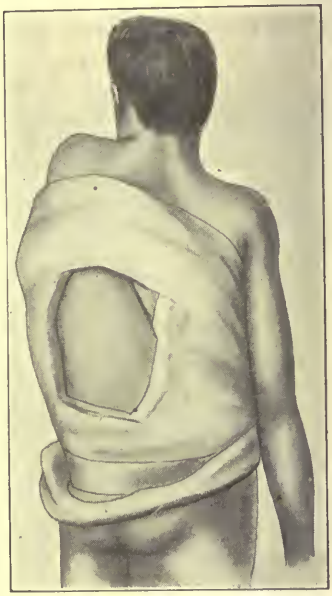


FIG. 671.—RIGHT SIDED THORACIC SCOLIOSIS IN "OVERCORRECTION" GYPSUM APPARATUS.

Contratorsion is prevented by the introduction of felt pads through the window.

Deformities of the Thorax.—A number of these are the result of developmental anomalies. Deformations of this sort interfere more or less with freedom of respiratory movements of the thorax and are not infrequently found in persons afflicted with lung tuberculosis.

CHICKEN BREAST.—In chicken breast (*pectus carinatum*) the thorax appears narrowed in its transverse diameter, so that the sternum protrudes beyond the level of the ribs. A depression is visible on either side of the sternum from the fourth to the eighth rib; the lower edges of the ribs are everted. The condi-

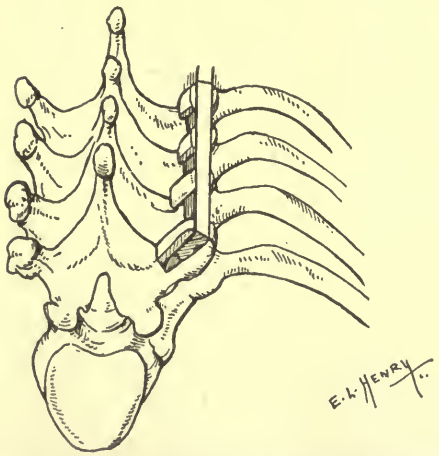


FIG 672.—BONE GRAFT IN TIPS OF LATERAL PROCESSES OF CONVEX SIDE OF CORRECTED PARALYTIC SCOLIOSIS (Albee).

tion is usually found in severe forms of rachitis in infants. It is frequently associated with rachitic kyphosis. As a rule, the deformity becomes less marked as life progresses. The *treatment* is similar to that of rachitis.

Funnel Breast.—Funnel breast (*pectus infundibuliforme*) is the reverse of chicken breast, the sternum being located behind the level of the ribs. It is regarded as the outcome of developmental anomalies. Spitzy[5] discusses its causation at some length. Lange[4] has succeeded in improving the condition by the use of suction over the funnel by means of a glass bell, from which the air is exhausted with a hand pump.

Cicatricial Deformities.—*Cicatricial deformities of the thorax* follow the shrinkage of the pleural cavity after inflammatory processes in this region. Much of this may be prevented by mechanical treatment of the causative process.

Spondylitis is taken up in connection with Surgery of the Spine, Part IX, chap. iii.

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CHAPTER VI

DEFORMATIONS OF THE UPPER EXTREMITIES

Congenital Elevation of the Shoulder.— Congenital elevation of the shoulder may be unilateral or bilateral. The condition was first described by McBurney and Sands[1] (1888) and was worked out by Sprengel[2] (1891). It is widely known as *Sprengel's disease.* In 1908 Horwitz[3] analyzed 136 cases collected from the literature. The lesion is characterized by deformation of the scapula and is regarded as the outcome of intra-uterine disturbances of development. Max Cohn[4] reports a case in which unilateral elevation of the shoulder was caused by tension of the levator angulae scapulae, the latter being due to scoliosis coincident with the presence of an adventitious vertebra in the thoracic region (7th).

The *symptoms* consist in an actual elevation of the deformed scapula. The scapula is rotated on its medial axis and its upper edge is luxated forward. The shoulder appears shortened and the entire scapula girdle droops forward and inward. The Röntgenogram reveals the deformation of the scapula, which presents exostoses and is frequently attended with a cervical rib and other bony anomalies. Congenital elevation of the shoulder is associated with scoliosis, the former being on the convex side of the latter. The deformity of the scapula interferes mechanically with lifting the arm.

Mechanotherapy, redressment, and apparatus do not improve the condition. Kölliker[5] resects that portion of the scapula which interferes with motion of the arm. If cosmetic effect alone is to be considered, section of the levator scapulae, supraspinatus, the anterior serratus and subscapularis muscles and subsequent fixation of the bone in the corrected position may be practiced. Spitzy[6] advises that a bony connection between the spine and the medial edge of the scapula be searched for in all cases. In some instances, an abnormally large coracoid process is responsible for deformity, this may be removed.

Acquired Elevation of the Shoulder.— Acquired elevation of the

shoulder may be regarded as an independent condition when it is not associated with deformation of the spine.

Its *causation* is varied. In most instances, the deformity is a part of a rachitic condition, but it is also ascribable to inflammatory shortening of the muscles that lift the scapula and to an hysterical basis.

The *symptoms* are similar to those of the congenital form and the *treatment* is the same.

Scaphoid Scapula.—Scaphoid scapula is described by Graves[7] as a clinical entity consisting of deformation of the scapula itself (scaphoid). The bone is very much elongated beyond the normal. The shoulder hangs forward and downward (winged scapula). Graves[7] regarded the condition as luetic in origin. However, subsequent investigation seems to prove it developed *in utero* from faulty position or as the result of an embryonic anomaly.

Rheumatic Shoulder.— So-called rheumatism of the shoulder has been made the object of careful study, especially by Goldthwait.[8] There is, of course, such a thing as rheumatism of the shoulder, which presents the usual clinical picture of this disease. However, the class of cases referred to exhibits persistent pain without the other manifestations of rheumatism.

According to Goldthwait,[8] the condition is characterized by forward and downward sinking of the shoulder, in which the pain is due to two separate anatomical peculiarities. One consists in compression of the bursa between the coracoid process and the minor tubercle of the humerus; the other, in pressure on the nerves of the brachial plexus.

In either event, relief is afforded by holding the shoulder in its normal position. This is accomplished by correctional gymnastics, though at times it becomes necessary to fix the arm for several weeks in the fully abducted position.

Obstetrical Paralysis of the Arm.— Obstetrical paralysis of the arm usually occurs sequentially to difficult labor, though it may develop in a normal delivery.

Two causative factors enter into its development, injury to the *brachial plexus* (Bieslaski[9]) with or without fracture of the humerus, and a *genuine distortion* (Lange[10]).

The *symptoms,* in recent cases, consist in limitation of the function of the joint; later, atrophy, contractures, internal rotation of the arm, and drop wrist appear. The *differential diagnosis* is established by the electrical reaction.

The *treatment* in fresh cases of distortion consists in fixation of the

arm in external rotation and abduction (90°) in a gypsum bed. Later, passive stretching of the capsule and active gymnastics are employed. If these measures fail, forcible redressment under narcosis and immobilization in the corrected position for six weeks are employed. This is followed by the use for six months of the gypsum

FIG. 673.— GYPSUM BED FOR THE TREATMENT OF DISTORTION OF THE SHOULDER IN INFANTS.

bed shown in figure 673. In children more than two years of age, redressment does not afford relief. In these cases *osteotomy* is necessary.

For the purpose, an incision is made through the outer bicipital groove, the periosteum is elevated, and the bone is divided transversely.

The lower fragment is rotated outward and the limb is fixed in this position in gypsum for six weeks.

In *genuine paralysis, neuroplasty* is indicated.

Spontaneous Subluxation of the Hand.— Spontaneous subluxation of the hand (Madelung's deformity, radius curvus, fork hand) is a luxation of the lower end of the radius with the carpus toward the palmar side, so that the lower end of the ulna stands out prominently in the dorsum of the forearm (Fig. 674). Gaugerle[11] attributes its occurrence to relaxation of the ligaments of the wrist joint. The condition is readily controlled by the prolonged employment of a suitably constructed celluloid or leather "restraining manchette."

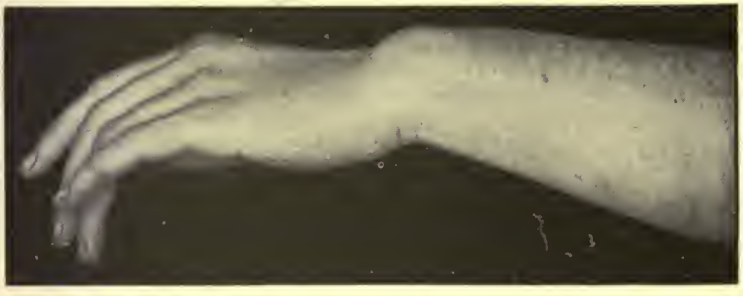

FIG. 674.— MADELUNG'S DEFORMITY (Whitman).

Dupuytren's Contraction.— This is already discussed in connection with Diseases of Joints (Part V, chap. vii).

Acquired Contractures.— Acquired contractures of fingers are taken up in Part IV, chap. ii.

Kruckenberg's Finger Contracture.— Kruckenberg's[12] finger contracture occurs in connection with luxation of the flexor tendons. Kruckenberg suggests that a groove be cut in the bone and the tendons placed in it. Spitzy[13] forms a flap from the fibrous covering of the metacarpophalangeal joint and turns it over the reduced tendons. He reports five cases successfully treated in this way.

Tendon Sheath Stenosis.— Stenosing tendon sheath inflammation at the styloid process of the radius is a lesion reported by De Quervain[14] and by Paulsen[15] as a clinical entity. It appears to consist in a chronic inflammation of the ligamentum carpi which strangulates the tendons; the latter are unchanged. Exposure by incision and division of the constricting bands is followed by prompt relief.

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CHAPTER VII

DEFORMITIES OF THE LOWER EXTREMITIES

COXA VARA

Coxa vara is an anatomical characterization of a bending of the neck of the femur, which produces a distinct clinical picture in many cases. It was first described by Fiorani[1] in 1882. The lesion may be divided as follows:

I. Congenital coxa vara.
II. Acquired coxa vara.
    1. Rachitic coxa vara.
    2. Static coxa vara.
    3. Traumatic coxa vara.
    4. Inflammatory coxa vara.
    5. Coxa vara from other causes.

Congenital Coxa Vara.— This is attributed to intra-uterine pressure and to diseases developed during fetal life.

The *clinical picture* of congenital coxa vara does not become manifest until the child begins to walk. It is usually attended with a club foot or ankylosis of the knee joint. *Abduction and flexion* at the hip are restricted because the neck of the bone is already abducted when the limb is held parallel to the head and the under side of the capsule is shortened, and also because the tip of the trochanter impinges on the border of the acetabulum or the ilium.

The *diagnosis* is made certain by the Röntgenogram. This is important, as the condition is readily mistaken for congenital luxation at the hip joint.

The *treatment* is taken up in connection with the acquired forms of coxa vara.

Acquired Coxa Vara.— The following varieties are seen:

Rachitic Coxa Vara.— Rachitic coxa vara does not occur as an *independent expression of rachitis,* being associated with X-legs, O-legs,

flat food, scoliosis, etc. Its local *symptoms* do not differ from those of the other forms of the deformity; it is usually bilateral.

It must be *diagnosticated* from the congenital form. This, according to Hoffa,[2] may be done by means of the Röntgenogram.

The tendency in rachitic coxa vara is toward spontaneous return to the normal.

Static Coxa Vara.— Static coxa vara means a disproportion between the weight bearing capacity of the neck of the femur and the load placed upon it. This occurs in young persons devoted to certain kinds of occupations which demand many hours in the erect position (waiters, gardeners, etc.). It is probable that the deformity occurs only in persons whose bones are deficient in lime salts.

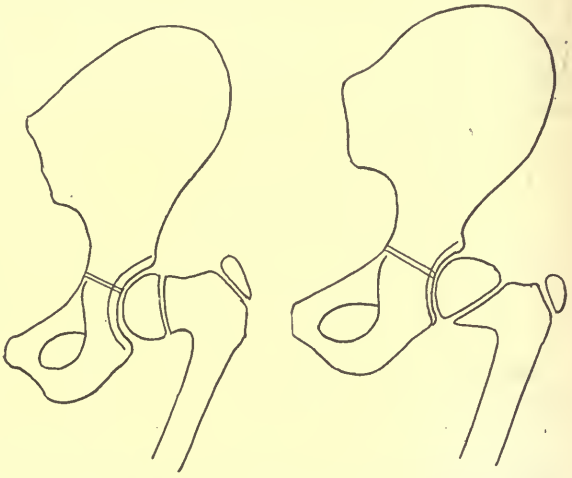


Fig. 675.— Schematic Presentation of the Head Epiphyses in Congenital (left) and Rachitic Coxa Vara (Hoffa).

Bade[3] devotes considerable space to the discussion of the changes in the head, neck and trochanter of the femur coincident to static coxa vara. He ends by stating that the neck is *always bent downward and backward* and that in come cases a partial luxation of the head is also present.

The *clinical course of static coxa vara* is characteristic. The patient usually seeks advice because of lameness with more or less pain in the knee and foot; *flat foot is a very common complication.*

Objectively, the most striking features are *restricted abduction, inward rotation, and the abnormally high position of the trochanter.* Extension of the limb is normal.

The *prognosis* is not unfavorable; healing with fair function often occurs. However, the disturbances are likely to persist for a long time.

The *treatment* during the painful stage demands *complete rest with extension.* The latter is important. If the Röntgenogram reveals

changes in the bone with deformity, correction under narcosis, stretching of the muscles, and the employment of the gypsum dressing is indicated. In severe cases, *bloodless infraction* may be employed. For the purpose, the pelvis is firmly fixed; the first abduction maneuver tears the adductors. Next, the fist of an assistant is forced into the groin and makes pressure against the head of the bone; the hands of the operator grasp the shaft of the femur near the trochanter and execute forcible abduction; a characteristic crushing sensation is imparted to the hands as the infraction occurs. The limb is placed in extension for six weeks. Lorenz[4] strongly recommends the method. Hofmeister[5] reports a number of failures.

Fig. 676.—Röntgenogram of Coxa Vara.

The *bloody operative measures of relief* are taken up in connection with Operations on Bones and Joints (Part IV, chap. vi).

Traumatic Coxa Vara.—Traumatic coxa vara is discussed in connection with fractures of the neck of the femur (Part IV, chap. vi).

Inflammatory Coxa Vara.—Inflammatory coxa vara results from infections of the neck of the femur, of which tuberculosis and infectious osteomyelitis are the most common. It also occurs as a part of arthritis deformans of indefinite origin.

Coxa Vara from Other Causes.—Coxa vara from other causes includes the cases in which the deformity is secondary to osteitis fibrosa, senile osteoporosis, the formation of bone cysts, etc. That is to say, any of the lesions common to bones may occur in the neck of the femur and be manifested by a more or less distinct clinical picture of coxa vara.

COXA VALGA

Coxa valga may be defined as the opposite condition to coxa vara, in which the angle of the neck of the femur to the head is increased and the shaft is near a straight line with it. According to Preiser,[6] the upper end of the femur is twisted forward and the acetabulum is distorted laterally. The condition is not common. Its underlying causation is similar to that of coxa vara, except perhaps that constitutional peculiarities are more often responsible for it than obtains in connection with the reverse deformity. On the other hand, Lange has observed coxa vara on one side and coxa valga on the other in the same person.

The *treatment* consists in massage, gymnastics, resistance movements, etc. Operative correction is rarely necessary.

GENU VARUM

Genu varum is the opposite of genu valgum (X-legs). The thigh and leg form a bow the convexity of which is outward and its apex corresponds to the level of the knee joint. In X-legs the anatomical cause of the deformity is fixed near the knee joint, while in genu varum it is located both above and below the joint, i. e., in the thigh and in the legs.

The participation of the femur and the bones of the legs is characteristic of genu varum and distinguishes it from the usual rachitic deformation of the bones of the legs alone, which is more properly designated as *crura vara.*

The *causation of genu varum* is similar to that of valgum, i. e., rachitis, exudative diathesitis, osteomalacia and congenital lues.

Genu varum is generally symmetrically bilateral. The degree of deformity is readily outlined with a pencil on a sheet of paper. Children with genu varum walk with a characteristic waddling gait, which the laity call "tying knots," "crossing toes," etc. As a rule, the condition disappears by itself.

The *treatment* is at first directed toward correction of the rachitic tendency. Although *local treatment* is not always necessary, most orthopedists place the legs in removable splints, which make correctional pressure and are worn at night. The employment of flat foot plates during the day is also helpful. If the deformity does not show a tendency to correct itself, *mechanical or manual modeling redress-*

ment, under narcosis, may be practiced. With a little patience this succeeds without actually fracturing the bones, the femurs and the bones of both legs being straightened at one sitting. When this does not succeed, bloodless fracture or infraction at the dome of the deformity is indicated. Bade[3] does this over a wedge, such as is used for reposition of congenital dislocation of the hip. In very severe

FIG. 677.— TYPICAL FULL BOW LEGS (Symmers' Bellevue Hospital case).

cases linear or cuneiform osteotomy is indicated. These are, of course, also executed at the highest point of the deformation. The principle of the operation is illustrated in connection with osteotomy for the relief of coxa vara (Part IV, chap. vi).

GENU VALGUM

Genu valgum, also known as knock knee, X-legs, and K-leg, presents A DEFORMITY OF THE LOWER EXTREMITY CHARACTERIZED BY ANGULATION, THE APEX OF WHICH CORRESPONDS TO THE KNEE JOINT. As to the

various theories of the *causation* of genu valgum, that of Mikulicz,[8] who attributes it to pathological conditions of the bones entering into the knee joint, is probably correct. The underlying cause in children is *rachitis* attended with an *irregular ossification of the epiphyses.* A second cause is *exudative diathesitis,* i. e., a constitutional osseous defect. This aspect of the subject is extensively discussed by Bade,[3] who presents a series of Röntgenograms showing the bony lesions responsible for the deformity; one of these, in which the genu valgum

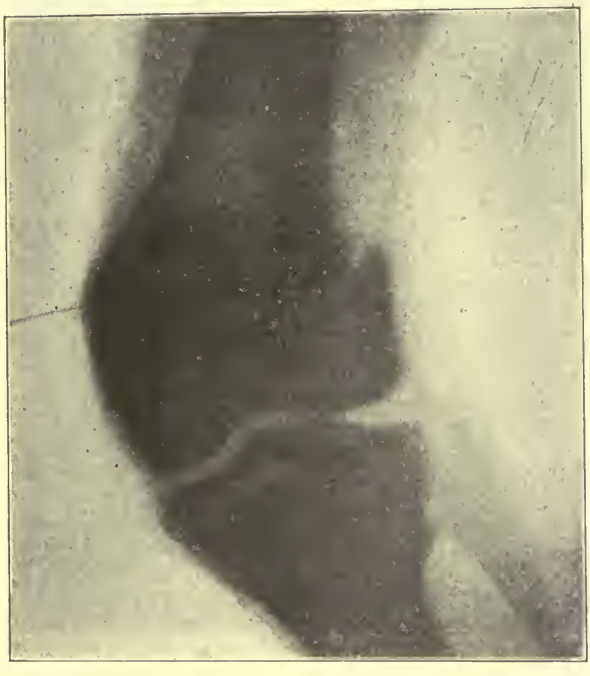


FIG. 678.— RÖNTGENOGRAM OF GENU VALGUM DUE TO CHANGES IN THE LOWER FEMORAL EPIPHYSIS.

is due to changes in the epiphysis, is shown in Figure 678. Additional valuable information is obtainable from the work of Lange[7] and of Kirmisson.[9] Bade[3] quotes Frölich as expressing the opinion that a certain number of the deformities are due to chronic staphylococcus osteomyelitis. It is certain that the *genu valgum of adolescence* is not satisfactorily explained on the basis of posture alone, and that an insidious chronic inflammatory process, such as Frölich speaks of, may well be the underlying cause.

The symptoms of genu valgum are clear enough. The knee joint protrudes inward. The degree of the deviation is easily made out by measuring the distance between the malleoli with the patient standing erect. With the patient lying down, the inner condyles of the two legs are apposed and an outline of the limbs is drawn on a sheet of paper. The deformity is often combined with bending of the tibia with the concavity inward. In genuine genu valgum the deformity disappears when the knee is flexed. The precise location and degree of the deformity is readily recognized in the Röntgenogram.

FIG. 679.— GENU VALGUM (Johnson).

The *treatment* of genu valgum relates primarily to the general care of the patient. This is exceedingly important.

The *local treatment* is divided into the *non-operative,* the *bloodless operative,* and the *bloody operation.*

The *non-operative treatment of genu valgum* consists in the employment of a night apparatus which is constructed over a gypsum model (p. 1337) of the leg (Fig. 680). During the day, the apparatus shown in Figure 681 may be worn; however, most orthopedists prefer that the latter be omitted. Spitzy[10] makes the children squat in the "Turkish position" for several hours each day and claims much for the measure.

The *bloodless operation* is really a modeling redressment carried out in one sitting under narcosis (Lange[7]). This stretches the external lateral ligament and molds the articular ends of the femur and tibia.

The method is followed by many failures. In its place, the epiphyseolysis practiced by Codevilla[11] is now much used. This consists in firmly fixing the femur down to the epiphyseal line on a hard table, the rest of the limb extending over the edge of the latter. The operator forcibly presses the leg downward until the limb is straight; this compresses the medial side of the epiphysis and causes its external side to gape away from the diaphysis. The limb is encased in gypsum from one and one half to two months. The results following the maneuver are good. Arrest of growth of the bone has not been observed.

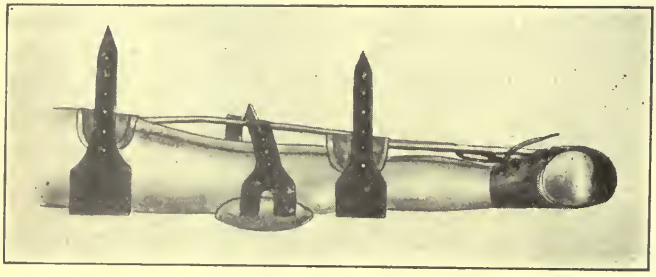


FIG. 680.—NIGHT SPLINT FOR CORRECTION OF GENU VALGUM.

The *bloody operative method of relief* consists in supracondyloid osteotomy, which is taken up in connection with Operations on Bones and Joints (Part IV, chap. vi).

GENU RECURVATUM

In genu recurvatum the knee is bent backward. In some cases the joint surfaces undergo changes caused by an effort to adapt themselves to the deformity (Delanglade[12]). The condition is ascribed to lack of room during *intra-uterine life,* though it may also develop later in life after *infantile paralysis,* as the result of *trauma* and of chronic inflammatory conditions of the knee joint (lues and tuberculosis) which weaken its capsule and ligaments. It occurs in the paralytic stage of tabes dorsalis.

This permits of a subdivision of the lesion as follows: (1) *Congenital;* (2) *paralytic;* (3) *atrophic;* (4) *osteogenetic,* the last being again divided into *traumatic* and *inflammatory.* In the latter the joint may become ankylosed in the faulty position.

The *treatment* is directed toward correcting the underlying cause. The employment of correctional apparatus, worn at night, is helpful. In severe cases arthrodesis may be necessary, while in cases of ankylosis

in a faulty position, partial or complete arthrectomy may be practiced (see Part IV, chap. vi).

Habitual dislocation of the patella is also taken up in Part IV, chap. vi.

DEFORMITIES OF THE FOOT

Flat Foot.— Flat foot is applied to a number of anomalies of the foot in which the *longitudinal arch is sunken* and, when "loaded" with the weight of the body, comes in actual contact with the floor (*pes planus*) (Fig. 682). This condition, by itself, is not common. It is not apparent when the patient is seated. Very often the first indication of the deformity appears when the foot, with the patient standing, is viewed from behind and an abnormal prominence of the internal malleolus becomes discernible (*pes valgus*).

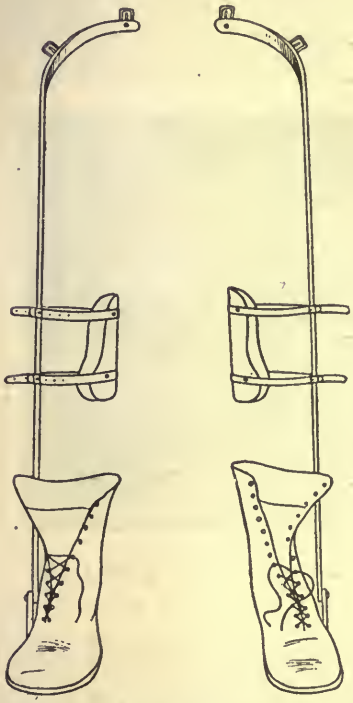


FIG. 681.— LONG BRACES FOR AMBULATORY CORRECTION OF GENU VALGUM.

When both symptoms—the obliquity of the axis of the heel and the flattening of the arch—are present, the condition designated as *pes planus valgus.* It is responsible for 80 per cent of the cases which consult the physician for relief of "pain in the foot."

In addition to this, a fourth deformation of the foot comes under consideration, *i. e.,* a *sinking of the transverse arch of the foot.* Normally, only the first and fifth metatarsal bones rest on the floor; when the transverse arch is sunken the second or third forms its lowest point; this is called *pes transversoplanus.*

The *causation of flat foot* is both *congenital* and *acquired.* The *congenital* flat foot is rare and is ascribed to lack of room in the uterus. the *postfetal flat foot* is due to a variety of causes, such as malleolar fracture, scar tissue contraction, etc. However, as a matter of fact, it is due, in the vast majority of instances, to *increased weight bearing.* In childhood it may occur in connection with *rachitis, malnutrition,*

anemia and *infectious diseases*. Of the direct causative factors, those relating to *habit of locomotion* and the *use of improper foot wear* are very important. There is no doubt that the tendency to evert the foot while walking with the view to being graceful, the enforced "toes apart and heels together" of military and school training, and the wearing of high heeled boots predispose to flat foot. It is the duty of parents to PERMIT CHILDREN TO BE "PIGEON TOED" AS LONG AS POSSIBLE. IF WAITERS, POLICEMEN, ETC., WERE TO TAKE DAILY "PIGEON TOE" EXERCISES THE PERCENTAGE OF FLAT FOOT WOULD BE GREATLY REDUCED.

The *objective changes* in flat foot are many and do not necessarily accord with the subjective disturbances. In addition to flattening of the plantar arch and the rotation in the long axis of the foot, the condition is attended with a *distinct protrusion of the sole of the foot* beneath the navicularis, which is due to a downward displacement of this bone. In this stage of the lesion comparison of Röntgenograms taken with the patient standing and seated is of great diagnostic value. Gradually the arch of the foot sinks more and more, the front of the foot widens and is elongated, so that the tendons are shortened and this leads to the *formation of hammer toes*.

FIG. 682.— PES PLANUS — FLAT FOOT (Johnson).

In the *fully developed flat foot the arch is entirely obliterated*. The skeleton of a foot deformed in this way is characterized by a complete change of architecture. The calcaneum is pronated and abducted, so that the fibular malleolus rests on the lateral edge of the former. The tuberosity touches the floor only with its median portion. The sustentaculum tali is broadened and moved backward. In the Röntgenogram the spongiosa of the bones of the tarsus is seen to be separated; normally, that of the talus and calcaneum cover one another. The *talus* has moved forward and inward; its articular surface is no longer horizontal in the malleolar fork, while the *navicularis, the three*

cuneiform and the cuboid rest on the floor. This causes changes in the relationship in the articular surfaces of the bones, which ultimately take on the form of a genuine *arthritis deformans,* with the formation of osteophytes.

Flat foot may be present for years without giving rise to symptoms, and suddenly, after a severe exertion, may develop an acute stage (*traumatic flat foot*).

The *subjective disturbances* are not in accord with the stage of the deformity. Changes of slight degree may cause severe symptoms, and not infrequently a complete flat foot is present without giving rise to discomfort. *Early pain* is usually present at *three points;* (1) at the inner edge of the sole in the course of the ligamentum tibiocalcaneo navicularis; (2) on the back of the foot over the joint between the talus and the navicularis; (3) on the outer side of the foot from the attach-

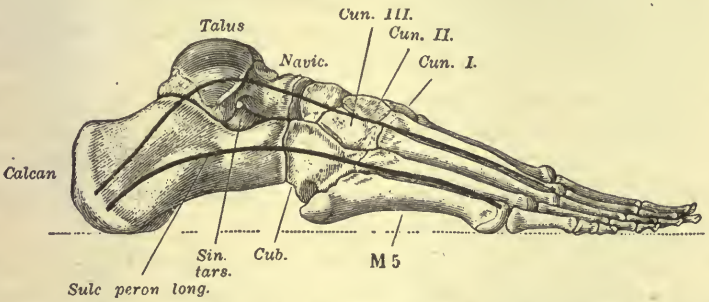


FIG. 683.— PROFILE VIEW OF THE SKELETON OF THE NORMAL FOOT, SHOWING THE ANTERIOPOSTERIOR ARCHES OF THE FOOT. (After Merkel.)

ment of the peroneus brevis to the external malleolus. In some patients the pain occurs along the entire length of the foot, apparently from straining of the medial interossei muscles, while others locate the pain at the heads of the metatarsal bones. Patients afflicted with flat foot are likely to "turn the ankle" while walking. Many of them present the clinical *triad,* (1) difficulty in "getting under way," (2) better when "under way," (3) worse after being "under way." *Very frequently, distressing pain occurs in the heel.*

Callus is often formed under the heads of the middle metatarsal bones. The sinking of the transverse arch is also attended with the localized "lightning pains" of *Morton's disease.* The pain is probably due to pinching of the plantar nerve as the arch gives way under the pressure of the superimposed weight.

After a time, the extra duties placed on the musculature of the leg

cause pain in the gastrocnemius muscles, and the tibia may also be the seat of dull pain. Pain in these parts may occur without pain in the foot.

THE TWISTING IN THE FOOT IN THE MALLEOLAR FORK MAY MODIFY THE STATIC ARRANGEMENT OF THE ENTIRE EXTREMITY AND PELVIS. Pain often develops beneath the patella and in the lateral ligaments of the knee; and the joint, in an effort to accommodate itself to the faulty position of the foot, undergoes compensatory deformation which, in severe cases, gives rise to arthritis deformans. A similar condition of affairs has been observed in connection with the hip and the sacroiliac joints (Preiser[13]).

The *diagnosis of flat foot* may be attended with considerable difficulty. It is not always easily separated from gout, rheumatism, and sciatica.

Above all, the surgeon must not assume that flat foot is not present because the footprint shows the presence of a plantar arch. As in almost all afflictions, the *history of the case is the most important determining diagnostic factor.* In most of the cases the "pain triad" spoken of above is present. In addition to this, the various symptoms presented under the head of subjective disturbances must be sought for. *The examination of the foot wear and observation of the patient's mode of locomotion are very important.*

Considerable assistance is furnished by the *footprints.* The normal outline is shown in figure 684A; B shows the print made by *pes planovalgus.*

The *treatment of flat foot* is of great practical importance. In the early stages of the lesion, massage, supination exercises with manual resistance, and the employment of properly fitting shoes will accomplish the purpose. The inner edges of the soles of the shoes may be raised.

As soon as the tendency toward valgus deformity appears, a properly fitting arch support is fitted into the shoe; this is not a simple matter. The support is best fashioned by an expert orthopedic technician. If necessary, the physician may prepare a gypsum model of the foot, in the manner indicated on p. 1337, and forward it to the technician. Care is necessary in providing support in cases of flattening of the transverse arch of the foot, especially when this is complicated by Morton's disease (see Whitman[14]). The use of the support does not lessen the need for the employment of foot gymnastics, which include the exercises of Ellis[15] and Roth,[16] and the use of the Zander

pendulum apparatus (p. 1341). Temporary relief may be obtained by the application of an adhesive plaster dressing.

In *obstinate flat foot* correction by redressment in narcosis is employed as follows: The foot is "wobbled" over a padded wedge until it can be fully supinated with one finger; it is then wrapped in cotton

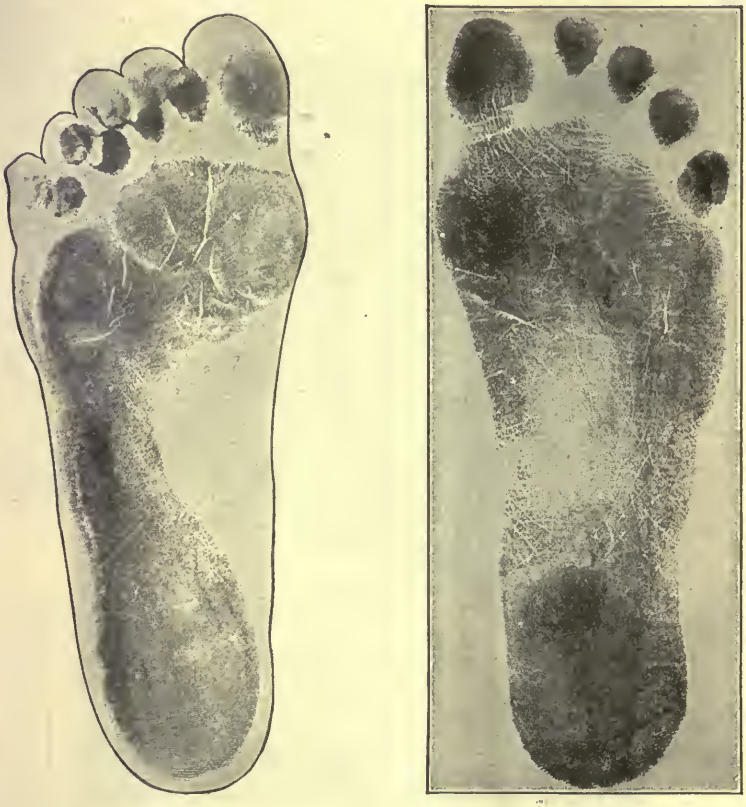


FIG. 684.— FOOT PRINTS. *A*, NORMAL PRINT. *B*, PRINT OF COMPLETE FLAT FOOT. Both arches have collapsed (Johnson).

and immobilized in gypsum (to the knee) in the corrected position. The patient is kept in bed for three days, at the end of which time he is allowed to walk in the gypsum cast. At the end of from four to six weeks the cast is removed and the usual after treatment is employed.

When arthritis deformans is established, redressment must be attained *by means of the osteaclast* (Fig. 634). In some cases subcuta-

neous section of the peronei tendons is necessary. This is not as successful as cuneiform osteotomy.

Cuneiform osteotomy for flat foot is not a difficult operation. Without regard for joints or ligaments, a wedge is cut out of the tarsus with a chisel and mallet at the site of the navicularis, the base of the wedge corresponding to the plantar and inner aspect of the foot. The wedge corresponds to about two thirds of the width of the foot; the

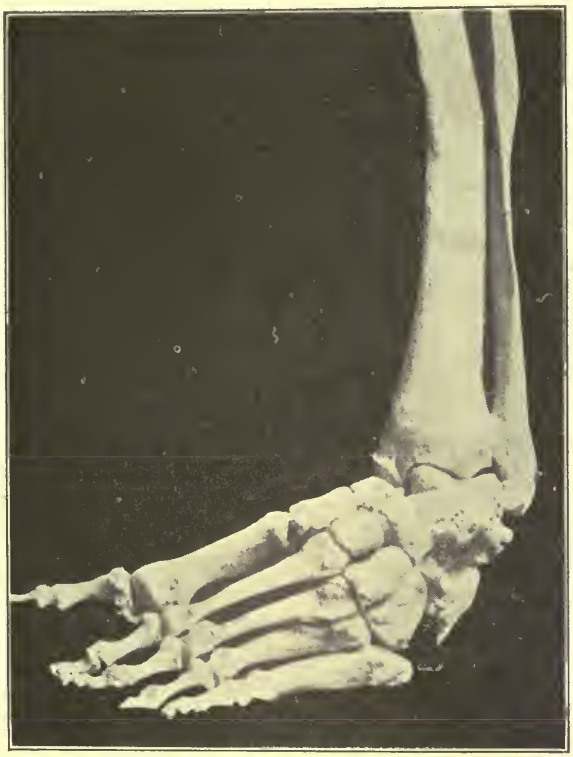
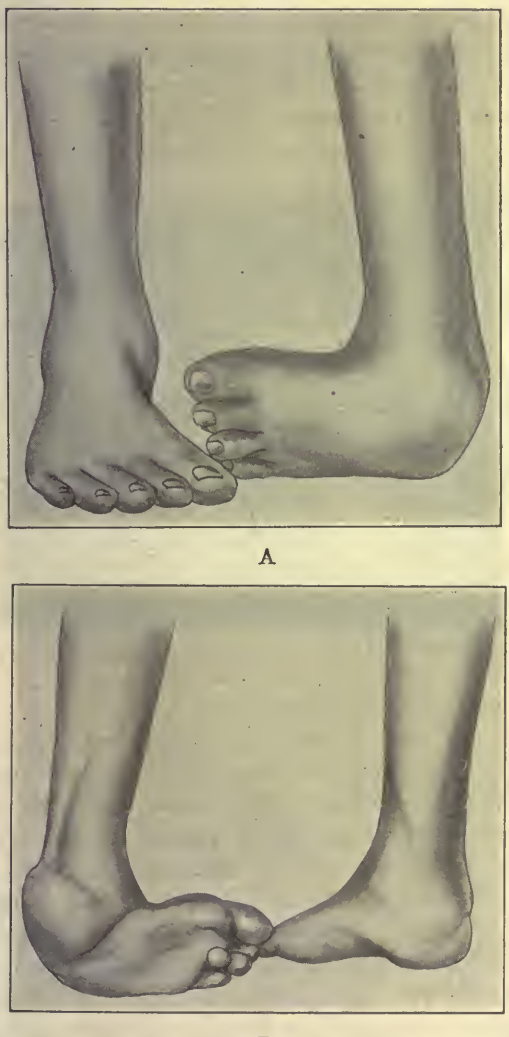


FIG. 685.—SKELETAL PREPARATION OF A CLUB FOOT IN AN ADULT (Lange).

outer third is cut through with the chisel. If approximation of the cut bony surfaces does not accomplish the desired correction, additional sectors of bone are removed. The wound is closed and the foot is encased in gypsum in the corrected position for eight weeks. The method gives excellent results. Both feet may be corrected at one sitting.

The *operative treatment of paralytic flat foot* consists in primary

bloodless redressment of the deformity and *secondary transplantation of tendons.* The paralyzed tibialis posticus is replaced by the flexor hallucis or the peroneus brevis and the tibialis anticus is replaced by the extensor hallucis. The execution of the technic requires considerable skill and a thorough knowledge of the anatomy of the parts.



A

B

FIG. 686.— SINGLE CONGENITAL CLUB FOOT.
A, Viewed from the front; *B*, Viewed from behind.

KÖHLER'S DISEASE.— Köhler[17] describes a condition observed in children that is characterized by lameness and is easily confused with flat foot. The Röntgenogram reveals shortening and widening of the navicularis. The density of the bone is also markedly increased. The absence of atrophy distinguishes it from bone tuberculosis. As the child develops the lesion undergoes spontaneous correction.

Club Foot.— Club foot is a *supination deformity of the foot.* The inner edge of the foot is adducted, the foot as a whole is rotated inward, and the sole of the foot is supinated. As a rule, club foot also exhibits a tendency to point and presents a certain degree of plantar hollow. This is called *pes equinovarus.* Club foot is either *congenital* or *acquired.*

CONGENITAL CLUB

Foot.— The causation of congenital club foot is ascribable to *heredity,* that is, club foot appears in generation after generation of the same family. It is also present in conjunction with spina bifida (p. 1426), etc. However, the chief cause would seem to be *abnormal intra-uterine pressure* from lack of room and amniotic bands. The influence of abnormal pressure is known because of the occasional presence of the scars of decubitus ulceration in the newly born. In addition to this, certain authorities consider intra-uterine poliomyelitis responsible for the congenital type of the deformity.

The *anatomical changes in congenital club foot* (Fig. 685) may be described as follows: The *calcaneum* is supinated and rests on its outer edge, so that its axis forms a bow with a lateral convexity; it is shortened and is in the abducted position. Its sustentaculum is underdeveloped, while on its lateral side the trochlear process, which gives passage to the tendon of the peroneus longus, is increased in size. The depression caused in the calcaneum by the increased tension of this tendon is deepened and is readily traced to the surface of the cuboid. The *talus* is also supinated; its axis is twisted outward (convexly), so that the outer side of its neck is lengthened and the inner shortened. The curve may be so acute that the tuberosity of the *navicularis* almost touches the internal malleolus. Indeed, in the adult neo-arthrosis is not infrequently formed in this situation, showing that the two bones have been in contact during weight bearing. The articular surface between the calcaneum and the cuboid is not perpendicular to the former, but is luxated inward. *The foot as a whole is rotated inward in the malleolar fork,* so that a portion of the body of the talus is luxated anteriorly. In the adult the joints show evidence of arthritis deformans and the deposit of osteophytes. The knee, the upper end

Fig. 687.— Adhesive Plaster Support for the Correction of Club Foot (Whitman).

of the tibia, and the condyles of the femur show deformations, the result of excessive internal rotation; and in the hip joint, the head of the femur undergoes a change in outline for the same reason. The *soft parts* on the inner side of the foot are shortened; the ligaments and the plantar fascia are shrunken. Of the muscles, the gastrocnemius, the tibialis posticus and anticus are contracted, while the ligaments, tendons and muscles on the outer side, especially the peroneus longus and the extensor digitorum, are much stretched.

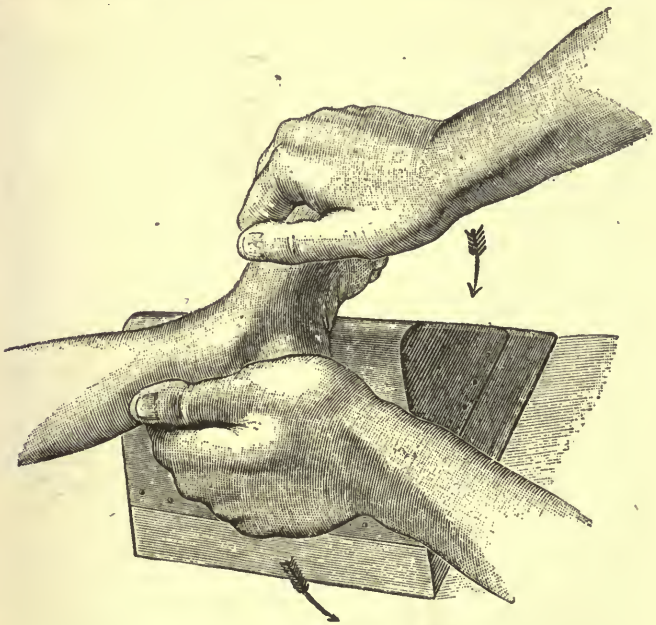


FIG. 688.— REDRESSMENT OF CLUB FOOT (Whitman).

Occasionally, congenital club foot is found in genu valgum. This peculiarity is possible only when the child is also rachitic.

The *clinical manifestations of club foot* are quite in accord with the deformity (Fig. 686). The tread of the foot corresponds to its lateral edge or even its back. Adventitious bursae and large calluses frequently form in these situations; the former often suppurate.

The *treatment of congenital club foot* is one of the most satisfactory attainments of modern surgery. It should begin within the first few days after birth, and at this time is a simple *manual redressment*. This consists in an attempt to "roll the foot up." The malleoli are grasped with one hand and the foot is sharply twisted so that the

plantar arch is flattened; at the same time the toes are forcibly elevated. This maneuver is carried out *several times daily*. Presently, *redressing adhesive plaster strips may be applied* (Fig. 687). The mother or nurse must be instructed to watch for disturbances of circulation. It is amazing how much may be accomplished by this simple means.

Redressment in narcosis is carried out as early as the fourth week of life, although most orthopedists wait until about the tenth month. It is performed as follows: The foot is postured with its dorsum (area of the cuboid) resting on the summit of a padded wooden wedge, such as is used for bloodless reposition of the hip. The one hand of the operator grasps the heel, the other, the metatarsus, and the foot is forcibly bent into the normal position until the plantar hollow has entirely disappeared (Fig. 688). When this is accomplished the toes are forced toward the shin as far as possible *and then* (*but not until then*) *the tendo Achillis is subcutaneously sectioned* (Preiser[18]). The small tenotomy wound is closed and the foot is postured as shown in Figure 689; or the leg may be at once placed in gypsum in the overcorrected position, as shown in Figure 690. *The child must not be taken from the table until the toes are rosy in color.*

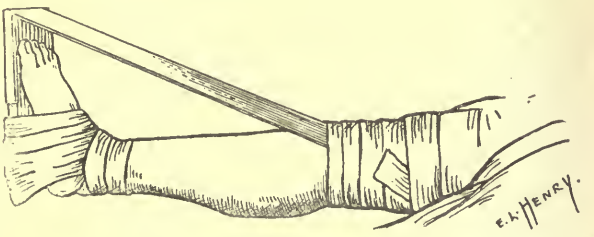


FIG. 689.— SAYRE'S APPARATUS FOR TALIPES EQUINUS.

The gypsum dressing is left *in situ* for several weeks and is renewed every four or six weeks for from one half to one year following the operation. This is followed by the use of an ambulatory apparatus, such as is shown in Figure 691; its mechanism is explained in the legend. As soon as correction is attained the limb should be frequently massaged, the apparatus being arranged so that this may be done. Apparatus, such as the one shown in Figure 691, must be worn for a long time (years). During this period correctional mechanotherapy may be used to advantage.

In older patients the problem may be attacked in the same manner as in children. However, redressment is best executed in two sittings,

the tendo Achillis being sectioned at the second one (v. Aberle[19]). In these cases the danger of fat embolism comes into consideration.

Of the so-called *bloody operations,* that of Phelps[20] and the radical cuneiform osteotomy have been abandoned in favor of the method of Ogston.[21] It is performed as follows: A curved incision is made over the talus and the greater part of its substance (except the articular surfaces) is removed with the gouge and the chisel. Eventually, the walls of the bone are forcibly "caved in" and the foot is placed in the

Fig. 690.— The First Application of Gypsum for Relief of Club Foot (Whitman).

corrected position. This is followed by immobilization in gypsum. The Röntgenogram shows that much of the bone substance is reproduced, but without deformity. The results of the measure are excellent.

Acquired Club Foot.— Acquired club foot follows *malleolar fractures, luxations of the talocrural or mediotarsal joints, osteomyelitis, and contraction of scar tissue.* It also occurs in connection with *poliomyelitis* (70 per cent) and *spastic paralysis.* The *treatment* varies with the cause. The traumatic forms are treated by *redress-*

ment, and, if necessary, *osteotomy is performed.* The *paralytic form* is corrected and this is supplemented by *tendon elongation* and *tendon transplantation.* In cases of total paralysis, arthrodesis, as indicated on p. 857, may be resorted to.

Pes Cavus — Hollow Foot.— Hollow foot, or claw foot (Fig. 692), is *a pathological increase of the plantar arch.* The deformity is rarely present alone and is usually complicated by *pes calcaneus or equinus.* The *congenital form* may coexist with *spina bifida* (also the occult

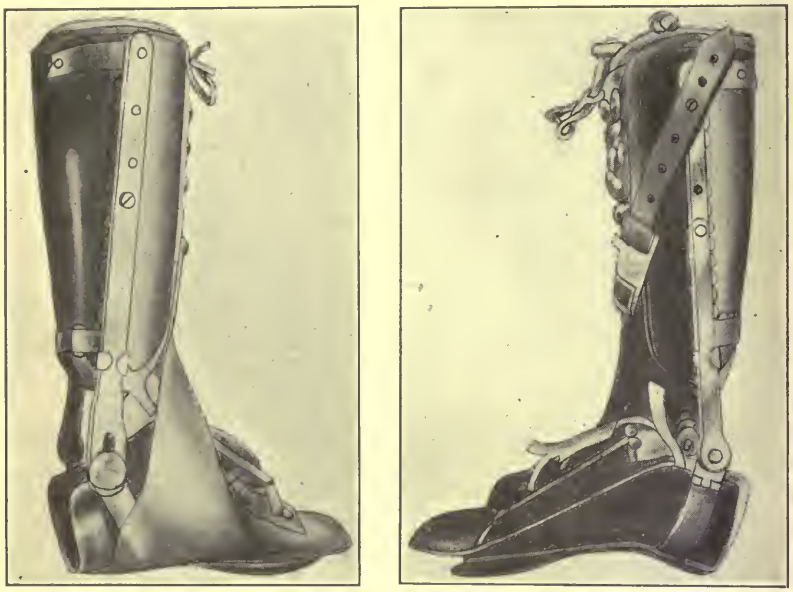


Fig. 691.— Ambulatory Correctional Apparatus for Relief of Club Foot.

A, The lateral foot hinge moves in a slot, so that the foot is forced toward the valgus position at each step. *B,* This is aided by a second hinge situated lower down. The apparatus is removed at night.

form). Its association with *chorea, myxedema* and *neurogenous contractures* of the plantar fascia seems to be established (Preiser[18]). The *treatment* consists in forcible redressment, as for club foot. In severe cases corrective cuneiform *osteotomy* of the navicularis is permissible.

Pes Equinus.— In pes equinus (Fig. 693) the deformity consists in an *abnormal plantar flexion of the foot.*

Its *causation* is *congenital,* i. e., it may result from intra-uterine

pressure. In most instances, however, it is *acquired.* The acquired form is divided into those resulting from *cicatricial contracture;* from *arthrogenous causes,* such as *gonorrhea, tuberculosis, lues,* and *polyarthritis;* the *traumatic* (following malleolar and tarsal fracture); the *neurogenous;* and the *compensatory.* In persons confined to bed for a long time it develops from pressure of the bedclothes. The *majority* of the cases are of *neurogenous origin,* being due to either spasms of the muscles of the calf (spastic paralysis, hemiplegia, etc.) or to flaccid paralysis of the anterior tibial group of muscles (anterior poliomyelitis). The *compensatory form* occurs in connection with shortening of the limb from various causes, including congenital luxation of the hip.

FIG. 692.— PES CAVUS.

As the *result of locomotion,* the sole of the foot over the heads of the metatarsal bones becomes calloused, and in some cases the patient walks on the dorsal side of the toes. The talus is luxated out of the malleolar fork.

The *treatment* of the *congenital form* is similar to that of club foot (p. 1401). In the acquired form, efforts at its prevention should be included in the care of the cause. In the paralytic form, correctional apparatus and the employment of prothesis is indicated.

FIG. 693.— COMPENSATORY PES EQUINUS, DUE TO SHORTENING OF THE LEG.

In the *severe forms,* redressment under narcosis and section of the tendo Achillis are employed. The latter may have to be supplemented by *tendon lengthening* and by *tendon transplantation.* In complete paralysis, *arthrodesis* of the ankle joint (p. 857) is justifiable. In the *compensatory form,* locomotion is aided by prostheses of various kinds. One of these is shown in Figure 694.

Fig. 694.— Sketch of Prosthesis for Compensatory Pes Equinus (Cork Inlay).

Pes Calcaneus. — Pes calcaneus (Fig. 695) is a rare deformity of the foot characterized by *excessive dorsal flexion.* According to Lange,[7] of 13,000 deformities of the foot, 7 were congenital and 31 were acquired cases of pes calcaneus. Preiser[18] saw 7 in 5,000 cases.

Pes calcaneus may be *congenital*

Fig. 695.— Pes Calcaneus (Whitman).

and is then due to intra-uterine pressure; in these instances it is usually a part of a talipes valgus.

The *acquired form* may be due to cicatricial contracture of the soft parts of the dorsum of the foot. However, it is usually due to *inactivity of the muscles of the calf.* The latter may follow injury to the tendo Achillis, but is more frequently the result of the paralysis coincident to *anterior poliomyelitis.*

Fig. 696.—Röntgenogram of Hallux Valgus.

When due to a spinal lesion the condition is readily studied. The deformity is manifestly the outcome of the traction of the unapposed activity of the dorsal flexors. When the paralysis of the muscles is not complete or their power is partially restored, the deformity is limited to a *pure pes calcancus.* However, if the gastrocnemius is completely paralyzed, the "tug" of the dorsal flexors and of the short flexors of the sole causes the deformity to become a *pes cavus.* The anterior portion of the foot sinks into extreme plantar flexion and the posterior maintains the calcaneus deformity; in this way a *pes calcaneo-excavatus* (Chinese foot) is formed. If the peronei muscles are preserved, these drag the already deformed foot into a valgus position, so that the condition must now be designated as a *pes calcaneo-excavatus valgus.* The latter is the usual deformity seen in cases of spastic and congenital pes calcaneus (Pürckhauer[22]).

The *treatment of pes calcaneus* varies with its etiology. In the con-

genital form the same principles are followed as are described in connection with club foot. The aim is at first of course toward overcorrection. In some instances *tendon plication* (p. 1048) of the tendo Achillis is necessary (Lange[7]). In the poliomyelitis cases tendon transplantation, using the peronei muscles for the purpose, is advisable. In total paralysis redressment and arthrodesis may be practiced.

Fig. 697.— Hammer Toes.

Pes Adductus, or Metatarsus Varus.— This condition is often associated with syndactylia and polydactylia. The hollow of the foot is much exaggerated and the first, second, and third metatarsal bones are strongly adducted. The deformity is due to contracture (neurogenous) of the extensor tendons and is regarded as congenital in origin (spina bifida occulta). The condition is readily corrected by means of manual redressment or by the help of the redresseur (Duncker,[23] v. Frisch[24]).

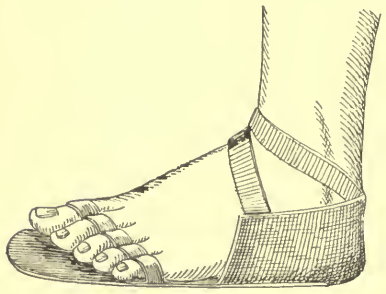


Fig. 698.— Sandal With Elastic Bands for the Treatment of Hammer Toes.

Hallux Valgus (Fig. 696).—This very common deformity is characterized by abduction of the great toe. It may be *congenital,* but usually is caused by *pointed shoes.* The position of the toe causes the head of the metatarsal bone to protrude, and, as the result of pressure (especially when a pes planus is also present), the bursa over the joint becomes inflamed and painful. Ultimately the joint itself develops a typical arthritis deformans, with the formation of osteophytes and joint mice. In the early stages proper adjustment of the boot is followed by relief. In most cases it is necessary to open the joint (by means of a curved incision) and remove the bony deposits (Wilson[25]). This relieves the pain, but does not correct the deformity; the latter is readily achieved by excision of the head of the metatarsal bone. After the wound is

closed the toe is maintained in the corrected position (in gypsum) for three weeks. Redressment of the pes planus may be executed at the same sitting.

Hammer Toe.— Hammer toes may be *congenital*, but are usually due to *short boots* or develop as a part of *flat foot*. The faulty position of the toe (Fig. 697) exposes it to undue pressure, with the result that "corns" and chronic bursitis make the life of the patient miserable. When several toes are affected, the apparatus shown in Figure 698 may be advantageously worn at night. Amputation relieves the situation, but is not practiced unless subcutaneous section of the flexor tendons and forcible redressment fails to correct the deformity.

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PART IX

REGIONAL SURGERY OF THE SPINE

CHAPTER I

GENERAL SURGICAL CONDITIONS

By regional surgery is meant the application of the general principles laid down in the first portion of this work to the various sectors of the body. For instance, the invasion of the vertebrae by the tubercle bacillus, the spirocheta pallida, or by pyogenic bacteria is governed by the same laws as obtain in connection with the other bones of the body, yet certain signs and symptoms are due to the location of the pathological process, and these must be met by special measures of relief. On the other hand, the simple disturbance of function in a given part does not express the character of the causative process, and this will escape recognition, unless the general laws are applied to the portion of the body under consideration. Deformity of, and pain in, the spinal column may be regarded as evidence of a disease of the spine, but its intelligent treatment rests on the establishment of the character of the causative process; this can be arrived at only by taking into account those principles to which so much space in this work has been devoted; it also explains why this has been done, and excuses, perhaps, the comparative meagerness of the space allotted to the surgery of special parts. The latter becomes comparatively easy when the subject matter of the general part has been mastered. It is manifestly impossible, in a work of this kind, to furnish a complete presentation of the various special branches of surgery. However, an effort is made to give the student and practitioner a basic, working knowledge to guide him in the field of his endeavor, and to so equip him that he will find less difficult the rest of the journey.

INJURIES OF THE SPINE

Injuries of the Spine Other Than Fractures and Dislocations. (For fractures, dislocations and fracture dislocations, see Part IV, chap. v.) —As stated in connection with the general considerations regarding regional surgery, special signs and symptoms are the outcome of

the involvement of special parts, and consequently these parts are subjected to special diagnostic measures. As applied to the spine, these measures relate to the *X-ray* and the *examination of the spinal fluid.* The technic of the former is of course not properly discussed here; aspiration of the spinal *canal* is, however, a surgical procedure, and as the characteristics of the spinal fluid have a bearing on almost all

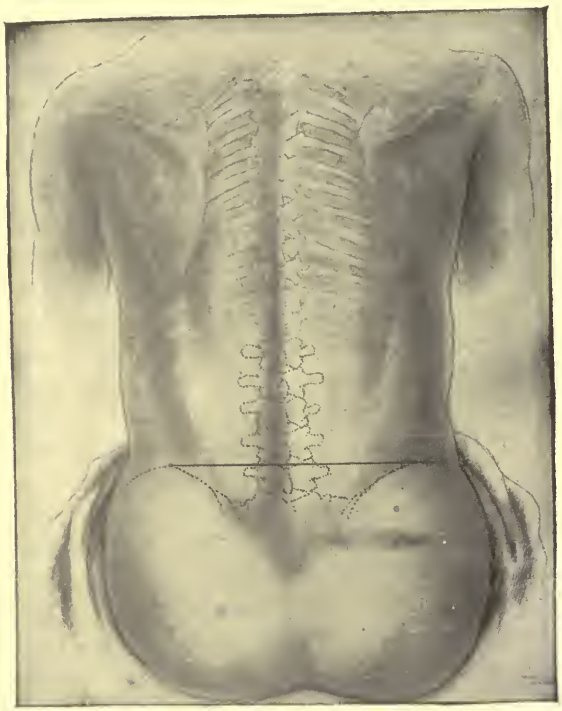

FIG. 699.—RELATIONSHIP BETWEEN A LINE DRAWN ACROSS THE ILIAC CRESTS AND THE INTERARCUAL SPACE BETWEEN THE FOURTH AND FIFTH LUMBAR VERTEBRAE, THE POINT OF ELECTION FOR LUMBAR PUNCTURE (Frazier).

conditions discussed in this part of the work, the technic of obtaining it is taken up at this time.

In *lumbar puncture* (rachicentesis) the needle must be introduced below the cord and between the strands of the cauda equina; therefore, it must be introduced between the second and the fifth lumbar vertebra; the most desirable of the avenues of approach here is the inter-

space below the third vertebra. A line projected from the summit of the crests of the ilia traverses, in most instances, the spine of the fourth lumbar vertebra (Fig. 699). The puncture is made in the

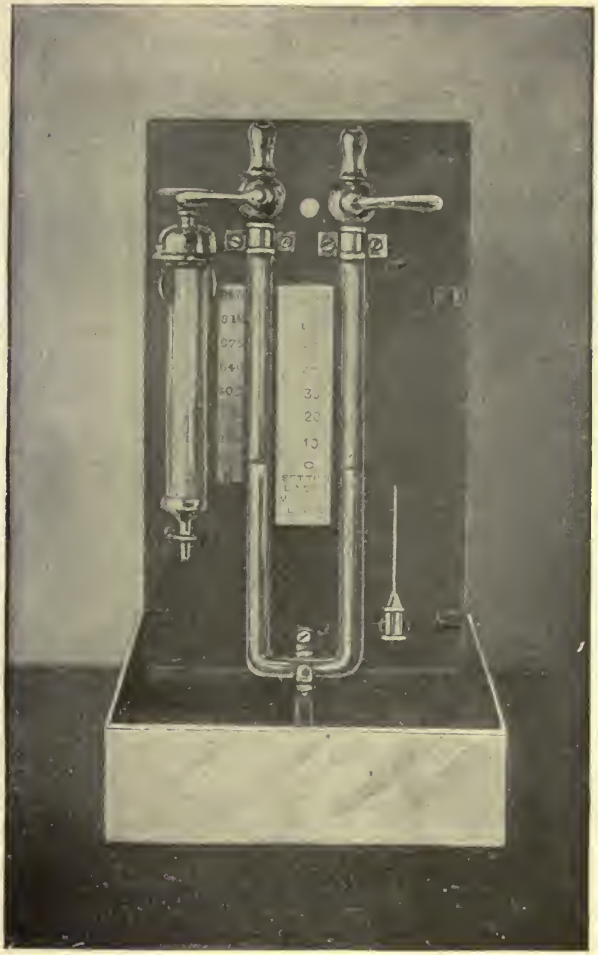

FIG. 700.— LANDON MERCURIAL MANOMETER (Frazier).

median line. The *equipment* for the purpose consists of a *manometer* for measuring the pressure of the cerebrospinal fluid, two sterile test tubes, a solution of collodion, an applicator, a needle (preferably platinum), a hypodermic syringe, and a solution of 5 per cent iodin.

The needle may be equipped with a stopcock. Frazier[1] thinks the Landon mercurial manometer (Fig. 700) meets the requirements.

Lumbar puncture may be performed with the patient in the *sitting or horizontal position.* In suspected brain tumor the latter is preferable; as a matter of fact, it should be employed in all instances. However, both positions are shown (Figs. 701-702). The skin is first punctured with a hagedorn needle, following which the hollow needle is introduced into the slit thus made and directed mesially and slightly upward; transfixion of the dura is recognized by the sudden absence

FIG. 701.— PROPER POSITION FOR LUMBAR PUNCTURE IN RECUMBENT POSTURE. Patient brought to edge of bed or table, limbs flexed to arch the lumbar spine.

of resistance. The stylet is withdrawn and the fluid allowed to flow into the graduate; 5 c.cm. is enough for diagnostic purposes. The intraspinal pressure is determined by connecting the manometer with the needle. Finally the needle is withdrawn and the puncture is sealed with collodion.

The changes in the spinal fluid are dependent upon the character of the inflammatory process, and may be said to be directly indicative in a number of lesions and helpful in others in a negative way. The technic of the laboratory examination is taken up extensively by Frazier.[1] In view of the comparative newness of the procedure, it may be advantageous to consult the works of the men quoted in the bibliography (end of chapter), of which those numbered from 2 to 54

inclusive refer to the subject. In this connection, the publications of Frazier[1] and Mestrezat[48] are perhaps the most complete (they also present extensive bibliographies), especially that of the former, whose text, being in our own language, may be the most valuable to the English speaking student. It is perhaps wise at this time to call attention again to the fact that aspiration of the spinal cord is not

FIG. 702.—LUMBAR PUNCTURE, SITTING POSTURE.

The patient leans forward, resting elbows on thighs in order to arch the back and widen the interarcual spaces. The needle is introduced in the interspinous space below a line drawn across between the crests of the ilia (Frazier).

without danger. This was brought out some years ago by Krause,[55] who observed two fatalities attendant upon the simple act of connecting the column of fluid in the canal with the manometer (that of Krönig), the patient being in the sitting posture. A similar unfortunate event occurred in the clinic of the writer in a case in which there was no lesion of the brain or cord, the puncture being made for

spinal anesthesia. The patient (a man of seventy) succumbed just as the fluid (5 c.cm.) was withdrawn, and before the anesthetic was injected.

The *after treatment* is important. The seemingly slight procedure is at times followed by severe headache (which may last a week) and vomiting. The patient must stay in bed for twenty-four hours and anodynes be given, if necessary. Aggravation of symptoms is reported by many authorities and has been observed by the writer in several instances. If more than 5 c.cm. are withdrawn, the quantity evacuated must be replaced with sterile salt solution.

Sprains or Distortions of the Spine.— These conditions are analogous to those of other joints; they may, however, involve more or less damage to the cord. Because of the great mobility of the cervical and lumbar segments of the spine, these are most liable to be the seat of injuries of this sort. The injury may result from a blow or a fall, the force being transmitted in the long axis of the spinal column. The mechanism is similar to that provocative of fracture or dislocation, only less in degree. The injury implies either overstretching or more or less extensive laceration of ligaments; transitory disturbance of the relationship of the vertebral bodies to each other may be assumed. The injury to the cord, seen at autopsy, without any dissociation of relationship of the bony parts in this situation, is explainable on this assumption. These injuries may be *contusion* (myelitis hemorrhagica) and *laceration* of the cord and pia, or *subdural hemorrhage,* or both.

The *clinical manifestations of sprains* include voluntary *restriction of motion, pain* on active motion, *tenderness* on pressure, and the *absence of evidence of fracture or dislocation.* The symptoms vary in accord with the severity of the injury. In severe cases the concomitant cord disturbances give evidence of the seriousness of the lesion. These may be limited to a slight *increase in tendon reflexes* or may include grave interference with motion, sensation, and the functions of the bladder and the rectum.

The *diagnosis* of sprain is *per force* based on the *Röntgenogram findings,* the only relatively accurate means by which the presence of fracture or dislocation may be excluded. The pain, the attitude of the head in sprains in the cervical region, and the absence of faulty alignment of the spinous processes and negative Röntegenological findings are to be considered indicative of sprain (Frazier[1]). The presence of blood in the cerebrospinal fluid (obtained by lumbar puncture, is helpful.

A *guarded prognosis* is necessary from the fact that an apparently trivial injury may stand ultimately in a causative relationship to distressing sequelae, such as *traumatic, chronic spondylitis* (Kümel's disease), with grave *deformity and permanent impairment* of function.

The essential of treatment is rest, at first in horizontal fixation, and later in immobilization (see spondylitis, Part IX, chap. iii). The mode of applying the treatment is determined by the gravity of the injury, as shown by the clinical picture. Active motion involving great effort should not be permitted until the tenderness has entirely disappeared.

Contusion of the Vertebral Bodies or the Ligaments.— This condition is more often suspected than demonstrated clinically. When the spinal column has been subjected to trauma and a complete examination, including the Röntgenogram and the findings in connection with the lumbar puncture, results negatively, the presence of a contusion may be assumed. Of course a sharp distinction between contusion and sprain is impossible.

A contusion, like a sprain, provokes pain on motion; tenderness is elicited by pressure over the injured area. The *Röntgenogram* is, as stated, helpful only in a negative way. The exposure would have to be a very successful one and the negative interpreted by an experienced eye to be considered as decisive in a diagnostic sense.

The *treatment of a contusion* of the spine must be carried out along the lines indicated in connection with sprain. *Fixation* should be persisted in until all the symptoms have definitely disappeared. A removable gypsum jacket may be worn in cases in which the symptoms are moderate in degree. *Massage* should not be employed until the acute symptoms have disappeared. On the other hand, *heat,* in the form of baking, relieves the pain, and, unlike massage, is without danger.

Fractures and dislocations are taken up in Part IV, chap. v.

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CHAPTER II

SPINA BIFIDA

Spina bifida, of the various *congenital deformities* of the vertebral column or spinal cord, is the *only one of clinical importance.* The term, which originated with Tulpius in the middle of the seventeenth century (Frazier[1]), is used in connection with THOSE CONGENITAL DEFECTS IN THE VERTEBRAL COLUMN THROUGH WHICH SOME OR ALL OF THE CONTENTS OF THE VERTEBRAL CANAL PROTRUDE IN THE FORM OF A CYSTIC TUMOR, varying in size from that of a hazel nut to that of an infant's head, and containing cerebral spinal fluid. As a rule, the skeletal *defect is in the arches* of the vertebrae, the tumor presenting posteriorly (*posterior spina bifida*); in rare instances, the *defect is in the vertebral bodies,* the tumor protruding anteriorly into the abdomen, pelvis or thorax (*anterior spina bifida*).

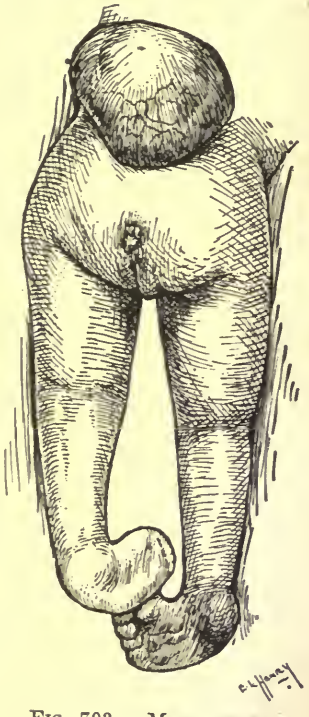

FIG. 703.— MYELOCELE: CLUB FOOT.

The *walls of the sac* may be composed of one or all of the membranes (*meningocele*), or of the membranes, together with the cord and roots, the cerebrospinal fluid being in the subdural or subarachnoid space (*meningomyelocele*), or the membranes and cord may protrude through the defect and the fluid may collect in the more or less dilated central canal (*syringomyelocele*). Therefore, in spina bifida the defect may be not only in the bone, but also in the cord and its membranes. The abnormal collection of cerebrospinal fluid is the important clinical factor.

The *congenital origin* of spina bifida is no longer questioned; the

pathological embryology is available in the works of v. Recklinghausen,[2] Muscatello,[3] Mouchet,[4] and others.

In view of the confusion with regard to anatomical types of spina bifida, the following *classification* (that of Frazier[1]) is submitted:

1. *Meningocele,* a protrusion of the dura, or both dura and arach-

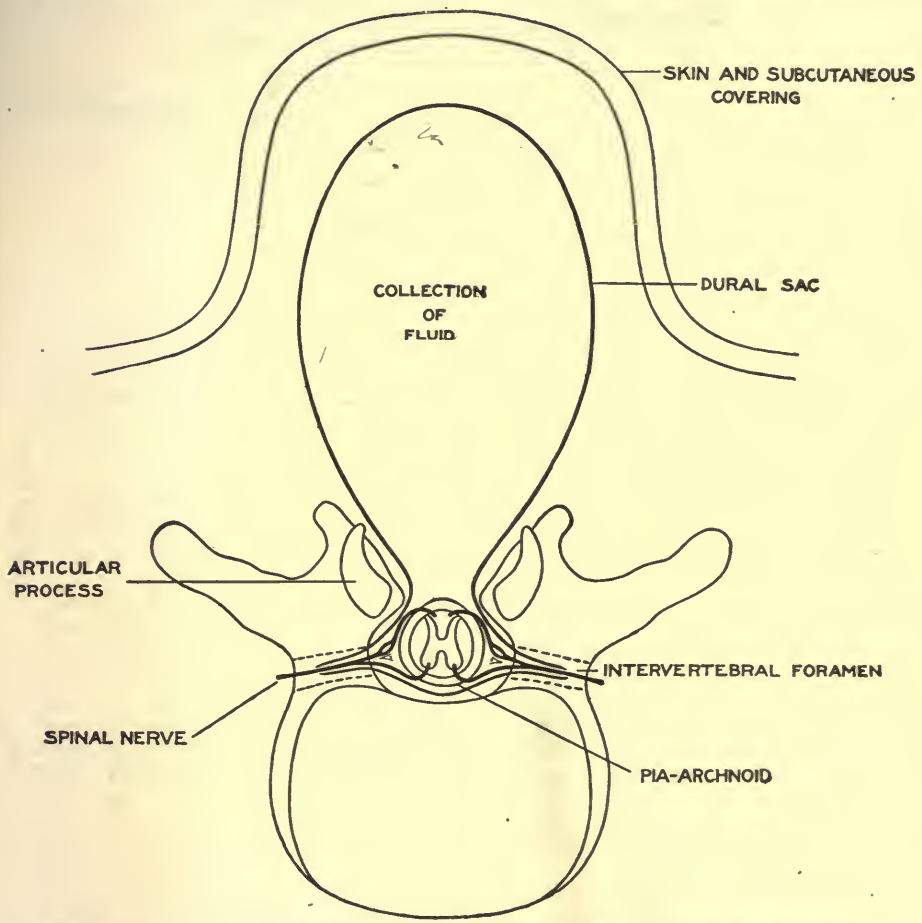

Fig. 704.— Diagram of Anatomical Relations of Meningocele (Frazier).

noid, through a cleft in the vertebrae, the collection of fluid being in the subdural or subarachnoid space posterior to the cord (Fig. 704).

2. *Spina bifida occulta,* really a variety of meningocele, except that the hernial protrusion is small, or hidden, or entirely lacking, and, therefore, on account of its slow development and the latency of its

symptoms, it demands special consideration both as to treatment and prognosis.

3. *Meningomyelocele,* a protrusion of the defective cord and anterior portion of the

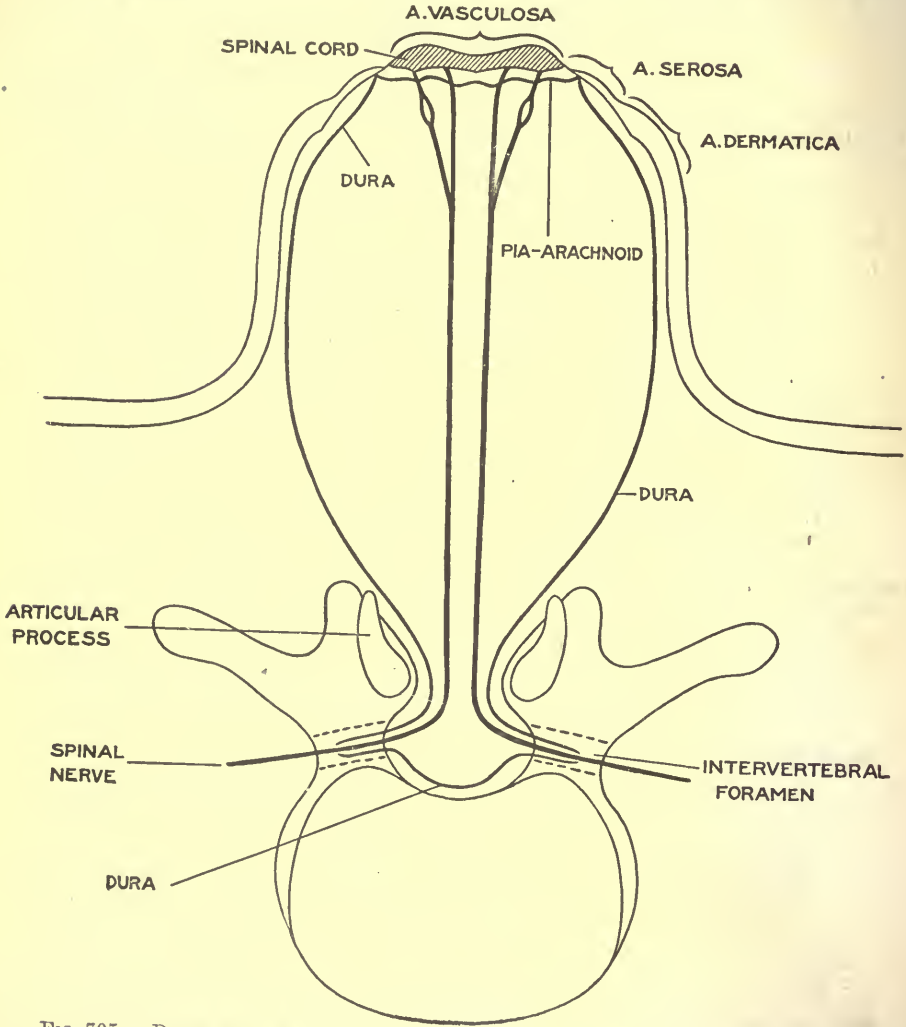

Fig. 705.— Diagram of Anatomical Relations of Myelomeningocele (Frazier).

vertebral column, the dura, the arachnoid, or the pia, the collection of fluid being almost invariably in the subarachnoid space, anterior to the cord (Fig. 705).

4. *Myelocystocele,* or *syringomyelocele,* a protrusion of the membranes and the cord. In this variety, however, the collection of fluid is in the central canal of the cord, which is greatly distended, the cord

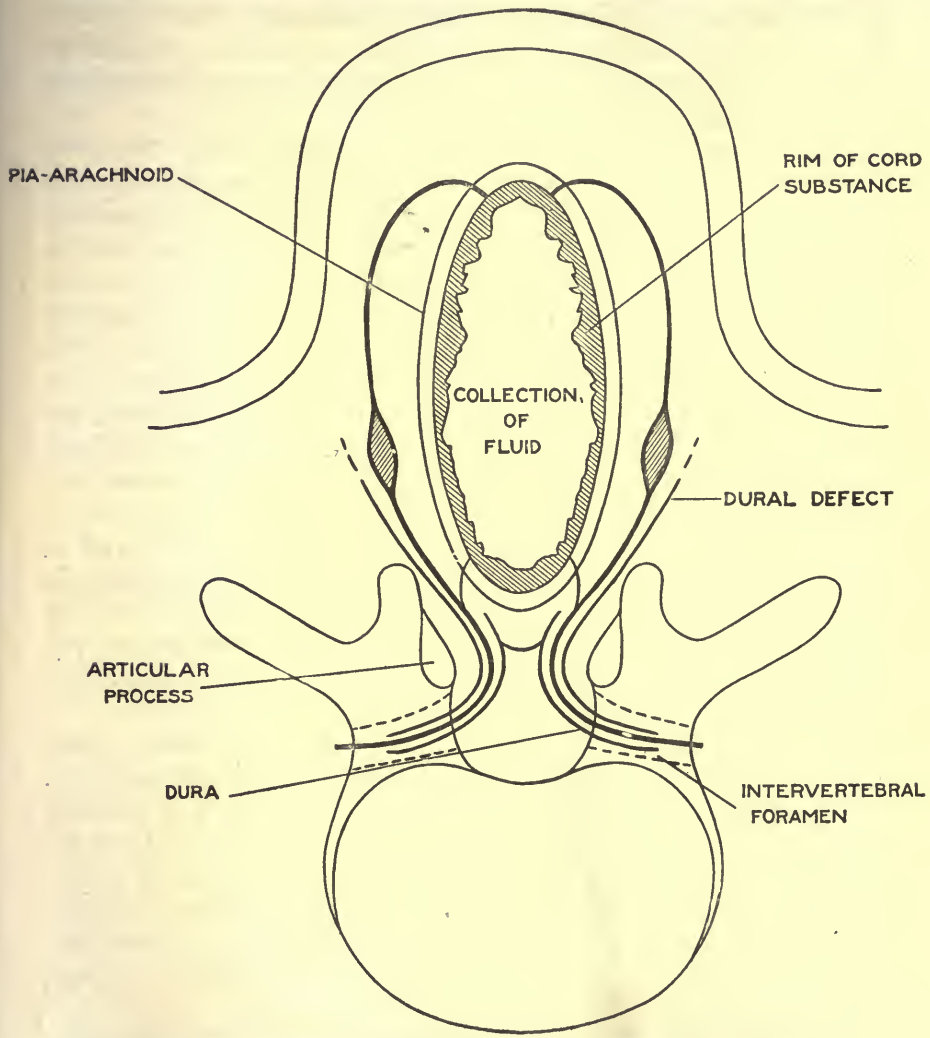

Fig. 706.— Anatomical Relations of Syringomyelocele (Frazier).

substance being atrophied and forming the inner lining of the sac (Fig. 706).

Spina bifida anterior is a rare condition. In most cases the *bodies of the vertebrae are defective,* allowing the contents of the spinal canal

to extrude, thus forming a cyst of the central nervous system, simulating a pelvic or abdominal tumor (Fairbairn[6]).

The *symptoms of spina bifida* fall naturally into three divisions, (1) the *local symptoms* due to the mere presence of the cystic protrusion; (2) the *nervous phenomena* caused by compression or degeneration of the cord, cauda equina, or nerve roots; (3) the symptoms due to *associated malformations and defects.*

Since the tumor is filled with cerebrospinal fluid and is in direct communication with the spinal and cerebral subarachnoid spaces, its quantity is variable. The size of the tumor is of course influenced by the position of the little patient, diminishing in size as the child is placed in the supine or elevated pelvic posture; its volume is transiently increased during forced expiratory acts such as coughing, sneezing or laughing. The degree of fluctuation depends upon the tension of the fluid and this in turn is dependent upon the dimensions of the defect. With a free communication into the spinal canal, the fluid may be forced into the canal; this is fraught with great danger of fatal cerebral compression. Frazier[1] reports that he witnessed an incident of this sort.

Inasmuch as the *spinal cord and roots are involved in all forms* of spina bifida *except the simple meningocele,* the *nervous phenomena* play an important part in the clinical manifestations. They consist in *motor, sensory, and trophic disturbances* and vary according to the location of the anomaly and the degree of compression or degeneration of the cord substance.

Of the cord symptoms, the motor are the most pronounced. These manifest themselves in the form of *complete paraplegia* with secondary contractures and paralysis of the bladder and rectum and terminal atrophy. *Talipes,* in one form or another, is an almost invariable accompaniment (Fig. 703). In addition to this, deformity of the vertebral column as a whole is not uncommon (Plagemann[7]). *Hydrocephalus* is a prominent feature in many cases of spina bifida, and anencephalus, absence of cerebellum, gliosis, diastematomyelia and syringomyelia are not rare (Frazier[1]).

Deformities and defects in remote portions of the body are not unusual; of these, congenital dislocation of the hip, syndactylia, polydactylia, polymastia, the absence of ribs, and herniae of various kinds are the most frequent.

Spina bifida occulta possesses *clinical significance* from the fact that, in addition to the external changes at the site of the defect, the *con-*

dition is associated with lesions such as hypertrichiasis, telangiectasis, pigmentations, and nervous disorders, *which often appear between the tenth and twentieth year of life,* when the membraneous band between the cord and the skin begins to compress or to make traction upon the spinal cord or nerve roots. These nervous symptoms consist of motor, sensory and trophic disturbances of the lower extremities and paralysis of the bladder and rectum. *Defects and malformations of other parts of the body* are very common with spina bifida occulta. Among these is diverticulum of the bladder (Pfanner[8]).

Spina bifida anterior, though not common, should be borne in mind *during operations for the relief of abdominal and pelvic tumors,* the origin of which seems to be obscure. As a rule, the condition is not discovered until the symptoms due to pressure of the tumor, such as pain, chronic ileus, obstinate constipation, frequency in micturition, etc., lead to exploratory exposure (Sawicki,[9] Tilp,[10] Robinson,[11] Neugebauer,[12] and Kroner and Marchand[13]). The presence of other malformations and of a dimple covered with hair in the sacral region may awake suspicion.

The *diagnosis of spina bifida,* except for the occult and anterior varieties, is comparatively simple; however, the protrusion must be differentiated from dermoids, sacrococcygeal teratomata, lipomata, and ischiatic hernia. On the other hand, the presence of a congenital hernial protrusion, rapid in its growth, situated in the median line, round or oval in contour, soft in consistency, fluctuating and varying in size in accord with the posture of the patient, should not leave much room for doubt as to its nature, especially when the defect in the spinal column is palpable. In *spina bifida occulta the diagnosis* is not easy. The Röntgenogram may be helpful and, as already stated, the coëxisting malformations may direct attention to the associated condition. The *anterior variety* is still more difficult of recognition. The Röntgenogram is of real value, as shown in a case reported by Kelly (*see* Frazier[1]).

In the *treatment* the selection of cases suitable to operative measures of relief is of prime importance. Frazier[1] thinks operation should be deferred until after the first year of life, unless rupture threatens, when it may be undertaken earlier. The contraindications to operation are hydrocephalus, coëxisting irreparable deformities, paralysis of the sphincters, and paraplegia. Hydrocephalus is regarded as a contraindication by Broca,[14] Muscatello,[3] Frazier[1] and others. The case of Kausch, quoted by Frazier,[1] in which the hydrocephalus was

taken care of by another operation, does not affect the general rule. As to paraplegia, the writer agrees with Hildebrand,[15] who says that

FIG. 707.— OPERATION FOR SPINA BIFIDA.

The dotted line around the neck of the sac represents the point at which sac is to be amputated (Frazier).

cases of this sort "should be left alone and should be allowed to die; in fact, the sooner they die the better."

Of the types, the *meningocele is the most favorable for operation. Spina bifida occulta* should be subjected to operation only *when symp-*

toms develop. Syringomyelocele may be operated upon when the tumor increases rapidly in size and is in danger of rupture. The *meningomyelocele* is the least satisfactory to operate upon.

In discussing the *treatment* of spina bifida, Frazier[1] states that in children who have reached the age of two months, in whom there is no paralysis or hydrocephalus and when the sac is covered with

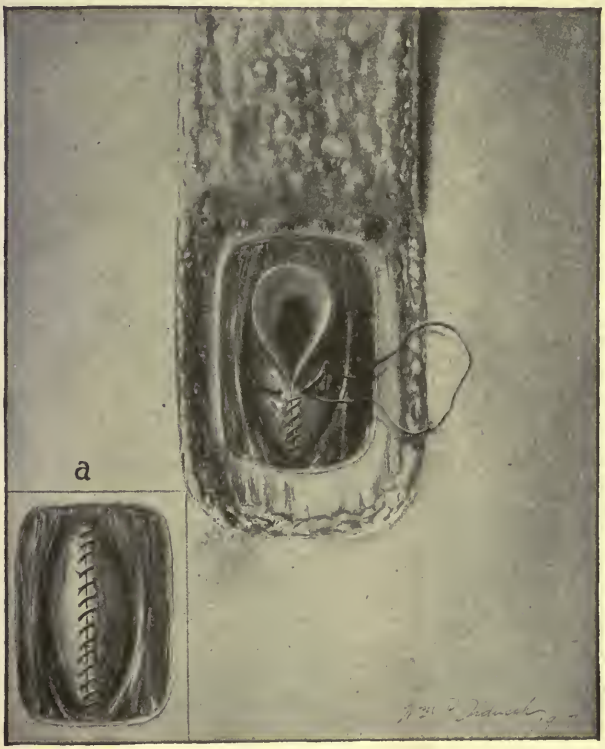

FIG. 708.— OPERATION FOR SPINA BIFIDA (continued).

The aponeurotic flaps are reflected and closed in the median line with a continuous catgut suture, *a* (Frazier).

healthy skin, the *injection of Morton's fluid* is justifiable, as it may accomplish the purpose and obviate the necessity for radical and more dangerous operative measures of relief.

The *operative methods of relief* include the use of musculofascial flaps (Bayer[16]), the transplantation of a flap from the fascia lata (Brodmann[17]), a periosteal flap (v. Bergmann[18]), the use of a bone

transplant (Zenenko,[19] Albee[20]), and fracture of the bases of the vertebral arches and transference of them to the median line (Babcock[21]). The method briefly described here may be regarded as indicating the principles involved in operative repair of the lesion. For elucidation with regard to the osteoplastic methods, the reader is referred to the

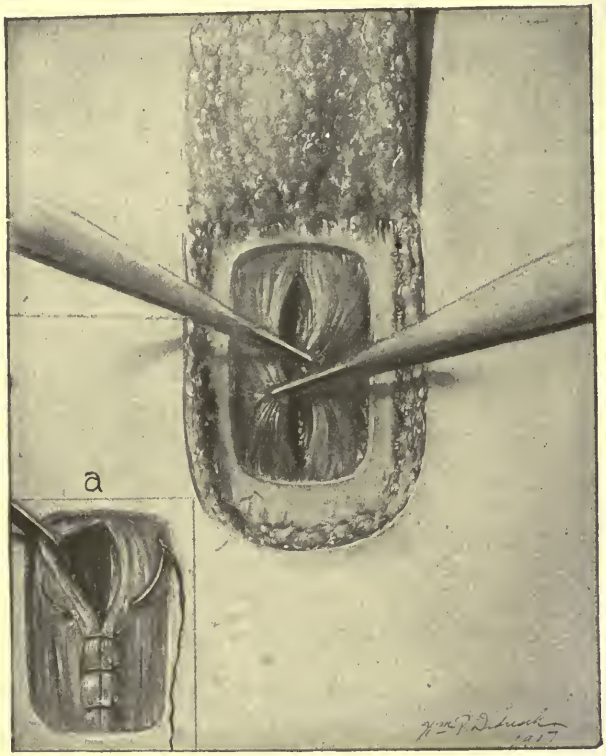

FIG. 709.— OPERATION FOR SPINA BIFIDA (continued).

The third layer in the closure of the defect is repaired by the mobilization of the muscles on either side of the defect, which are secured in the median line with interrupted catgut sutures, *a* (Frazier).

bibliography as stated above. Frazier's[1] method (which he states is generally credited to Bayer[16]) may be described as follows:

A U-shaped flap is dissected up and the cyst enucleated, as shown in Fig. 707, which also shows (dotted line) where the sac is amputated; the opening in the sac is closed with catgut. An incision is made in the sheath of the erector spinae muscle around the margin of the

hernial orifice, extending 2 c.m. above and below it, liberating two lateral flaps, which are reflected over the hernial opening and approximated in the center with a continuous catgut suture (Fig. 708). If the muscle cannot be approximated without undue tension, vertical liberating incisions are made on either side of the hernial opening. The muscle is then approximated in the manner shown in Fig. 709. Next, the vertical arms of the U-shaped incision are extended upward; a flap is dissected from the aponeurotic covering of the muscle and this is turned down over the site of repair (Fig. 710) and held in place with interrupted chromic gut sutures. Finally, the skin flap is replaced and held in place with silkworm gut sutures. The operation may be performed through a single, vertical, median incision. If the parts are badly nourished, a free fat and fascia transplant from the fascia lata may be used in place of the one turned down from above the defect. In *myelomeningocele* the sac is opened by a transverse incision, a portion of it is excised and the roots and ganglionic tissue returned to the canal.

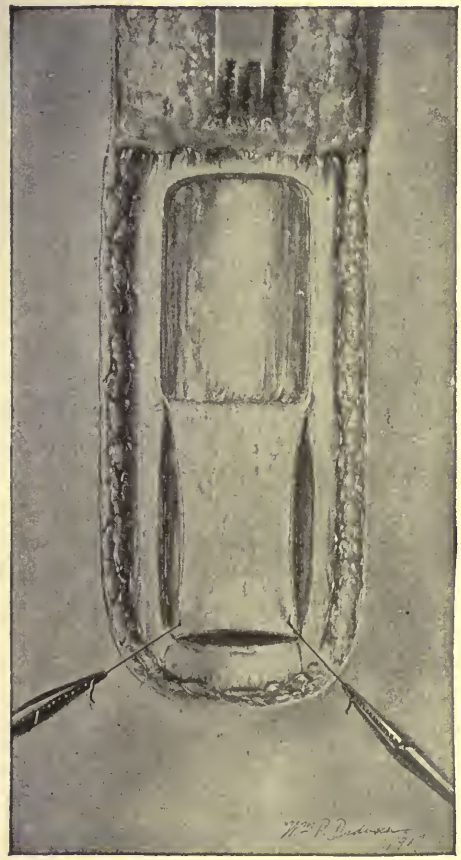

FIG. 710.— OPERATION FOR SPINA BIFIDA (continued).

The muscle layer is reënforced by the reflection of a U-shaped aponeurotic flap from above, secured in place with interrupted sutures (Frazier).

Regarding *operative prognosis,* Frazier[1] places the mortality at about thirty per cent.

In the *after treatment* the proximity of the operative field to the anus calls for the exercise of great vigilance as regards accidental infection.

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CHAPTER III

SPONDYLITIS

POTT'S DISEASE is a term applied to the MANIFESTATIONS INCIDENT TO DESTRUCTIVE CHANGES IN THE BONES AND JOINTS OF THE VERTEBRAL COLUMN. The term has been obtruded into the literature since 1779, when Pott first described an affliction of the spinal column attended with deformity, pain, and paralysis. At his time and, indeed, for a considerable period of time after him, the character of the causation was not understood. For many years all destructive conditions in this situation were regarded as tuberculous in character and, as a matter of fact, most of them are; however, a larger number of them are due to infections of other kinds — lues, pneumonococcus, typhoid bacillus, pyogenic bacteria, etc.— than is generally believed even today. The term "Pott's disease" should disappear and a terminology be employed indicative of the kind of process involving the bones and joints, such as tuberculous, typhoid, pyogenic or luetic spondylitis.

When *spondylitis* as a whole is viewed from this standpoint, it becomes patent that *the changes which take place in the bones and joints of the vertebral column do not differ histologically from those incident to the invasion of a similar provocative factor in the other parts of the body,* except perhaps inasmuch as the blood supply varies from that of other parts (Part II, chap. x), a conception which should rob the problem of some of its complexity and materially aid the student in understanding the pathological processes in the situation under consideration.

Farther than this, the general *symptomatology* of inflammatory processes in the vertebral column is the same as that attendant upon similar lesions in the other bones and joints, and this is also true with regard to the *diagnosis and the general principles of treatment.* There remain, then, to be considered only the *special symptoms* — the outcome of the *site of the lesion,* the *special diagnostic measure* to be addressed to it, and the *special* efforts at relief demanded.

The *special symptoms* of spondylitis relate to *pain, loss of function,*

deformity, and *secondary complications,* the latter being divided into abscess formation and *paralysis.*

The *pain* of spondylitis is *rarely localized* in the region of the dis-

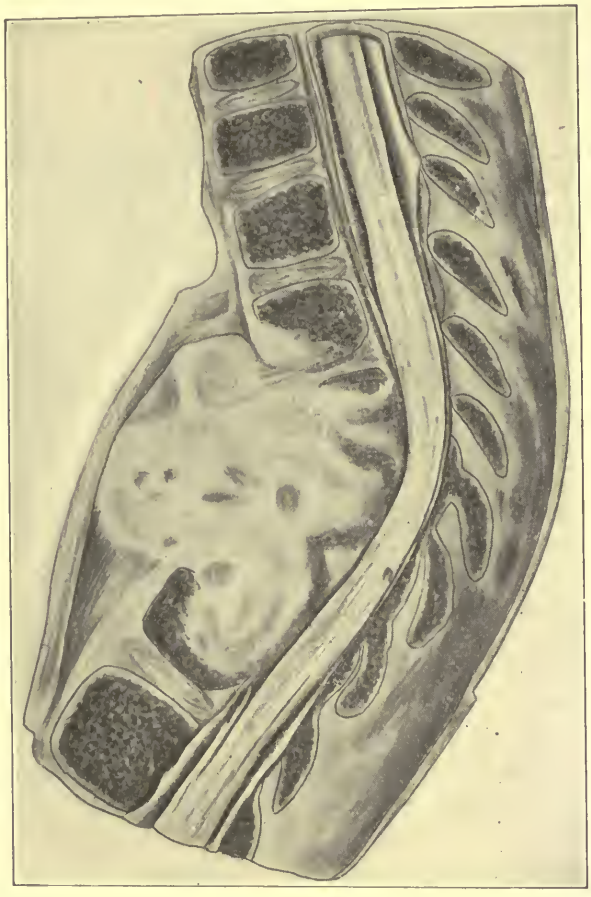

FIG. 711.— SPONDYLITIS IN THE LOWER DORSAL REGION.

Six vertebrae are partially destroyed. The abscess has lifted the anterior ligament and is making backward pressure on the cord. The cord is flattened. Complete paralysis was present.

ease, but is *referred to* the front and sides of the body, to a location in accord with the site of the lesion, such as the side of the *face, the neck, the thorax, the abdomen,* or the region of distribution of the *sciatic nerve.*

Impairment of the function of the vertebral column is expressed by restriction of voluntary movements of the limbs, or of the motility of the spine, due to contracture of muscles in an effort to assume a posture which relieves pressure, and because of reflex spasm.

Deformity (Fig. 711) is fundamentally the expression of changes in the form of the diseased bones and the intervertebral joints. The deformity may present an *angular projection* (kyphos) or may consist simply in *lateral or anteroposterior distortion.*

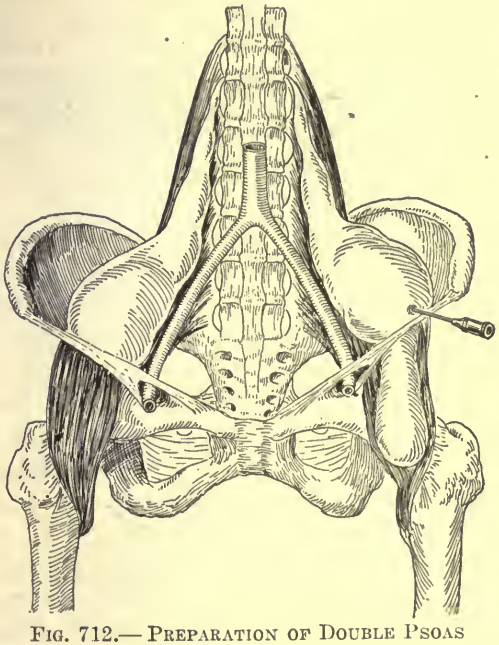

FIG. 712.— PREPARATION OF DOUBLE PSOAS ABSCESS.

The needle has punctured the cavity on the left side above Poupart's ligament.

Of the *secondary symptoms, abscess* (Fig. 712) possesses special interest because of the liability of the exudate to gravitate to a distant point. *Paralysis* is usually a late symptom and is gradual in its onset, unless a pathological fracture or dislocation occurs, when it may appear suddenly. It is the result of various causes. Of these, compression of the cord from *narrowing of the spinal column* is rarest. The pressure of *epidural exudate* and peripachymeningeal *granulations* and the *accumulation of pus* is more common. *Circulatory stasis* and the formation of thrombi is not rare. According to Lange,[1] local *anemia and edema,* due to the circulatory disturbances coincident to the malposition of the muscles and bones (in kyphos), may cause motor and sensory paralyses in restricted zones. In addition to this (in some instances), a certain area of the medulla of the cord may degenerate, and this, in turn, may be followed by ascending or descending degeneration (gliosis) of its columns. Early relief of pressure (mechanical correction of malposition, aspiration, or laminectomy) is followed by return of function unless sclerosis has *occurred.*

In the *lumbar region* the deformity is usually that of *lordosis,* i. e., a prominent abdomen, a hollow back. *Spasm of the psoas muscle* is an early symptom. *Pain* radiates over the *inguinal region and down the thighs.* In *abscess formation,* the exudate may gravitate to the pelvis (pelvic abscess) or a typical *psoas* abscess may occur. In the latter, the exudate makes its appearance just above Poupart's ligament (Fig. 712).

In the *thoracic spine* the deformity varies from so-called *"round shoulders"* to a well marked *kyphos* (Fig. 711). *Pain* is referred to the *abdomen* and radiates over the *sides of the thorax;* it is often spasmodic in character and is attended with *labored respiration* and *cough.* In *abscess formation* the exudate may appear between the ribs or posteriorly beside the spinous processes.

In the *upper region of the spine,* which includes the cervical and upper two dorsal vertebrae, the attitude of the patient is characteristic; the head is held rigid and the eyes follow moving objects; the chin is often held by the hands. These symptoms are especially marked when the lesion is high up. *Pain* is referred to the *ears* and *pharynx.* In the lower segment of this region *pain* is referred to the *neck,* the *sternal region,* and down the *arms.* In *abscess* formation the exudate gravitates to the *pharynx* and to the *neck,* where it appears in front of (rare) or behind (common) the sternomastoid muscle; it may appear over the posterior aspect of the vertebrae.

The diagnosis in general is based on the regional symptoms stated and on the faulty alignment of the spine as a whole. To determine the character of the process, the history of the case, the development of the clinical picture in connection with other possible causative processes, such as pneumonitis, typhus abdominalis, lues, that omnipresent tuberculosis, rheumatism, gonorrhea, etc., the acuteness or chronicity of the process, the *Röntgenogram,* and the *examination of the cerebrospinal fluid* must be taken into account. However, the reader is again reminded that *the process is not always tuberculous,* despite the evidence of tuberculosis elsewhere in the body. The *serological examinations* (lues, gonorrhea, etc.) should not be omitted. Of course a patient with lues may develop a tuberculous spondylitis.

The *Röntgenogram is of great diagnostic value;* however, the negative must be properly interpreted, which is not always easy, especially with regard to the character of the lesion. Fig. 713 shows a Röntgenogram of a case of tuberculous spondylitis in the lumbar region. The Röntgenogram is also of value in recognizing the presence of *gravita-*

tion abscesses. In some instances it will reveal the presence of the thickened pyogenic membrane, and, when a fistulous tract is present, this is made visible by the injection into it of Beck's[2] bismuth paste.

When the *presence of purulent exudate* in the tissues surrounding the spinal column *is suspected, the employment of the aspirating needle for diagnostic purposes is justifiable.* The possibilities in this connection do not seem to be sufficiently strongly emphasized, not alone as to diagnosis, but also with respect to measures of relief. For the purpose, the aspirator shown in Fig. 364 or a large sized suction syringe connected with a hollow needle may be used.

FIG. 713.—FISTULOUS TRACT CONNECTING WITH LUMBAR SPONDYLITIS MADE VISIBLE IN THE RÖNTGENOGRAM BY INJECTING BISMUTH (Lange).

The *treatment of spondylitis* is based on the principles laid down in the discussion of inflammatory lesions of bones and joints (Part IV, chap. iv). *In acute inflammatory processes* the logical procedure would be to expose the lesion and excise or drain it in the same way as in acute osteomyelitis or arthritis elsewhere in the body. Unfortunately the location of the process makes direct attack a difficult problem. However, it is fair to assume that, as our methods of diagnosis become more dependable, or, it may be said, as those we possess are more widely employed, it will become feasible to expose the lesion early in the disease and to apply direct measures of relief. In cases of *tuberculous spondylitis* with paralysis *decompressive laminectomy* has been followed by excellent results (p. 1472).

When the presence of pus is established, it should be drained as early, and as near the seat of the lesion, as possible, with the view of obviating gravitation of the products of inflammation to a distant point and secondary invasion *en route.*

The *general treatment of spondylitis* is important. Since in most instances the lesion is tuberculous, the general treatment is similar to

that employed in combating this disease in other portions of the body. It must be remembered that in not a few of the cases the lesion is luetic, gonorrheal, rheumatic, etc.

Just as in bone and joint lesions elsewhere in the body, the *mechanical treatment of spondylitis* aims at immobilization of the afflicted

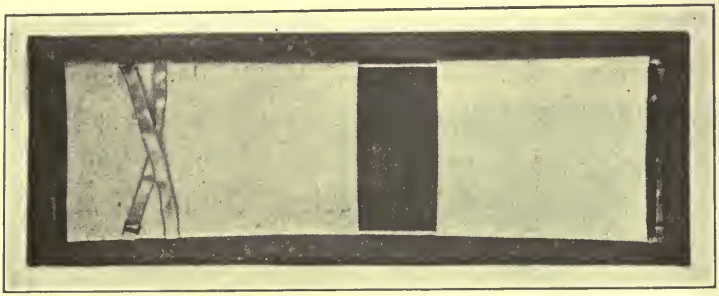

FIG. 714.— BRADFORD AND LOVETT'S BED FRAME FOR HORIZONTAL IMMOBLIZATION IN CASES OF SPONDYLITIS (Whitman).

part in such a manner that healing may take place with a minimum of deformity and permanent impairment of function. For the purpose, apparatus of various kinds are employed. These must of necessity vary in construction in accord with that portion of the vertebral column involved, *the aim being* fixation in a position in which the strain of use and the pressure of the superincumbent weight of the

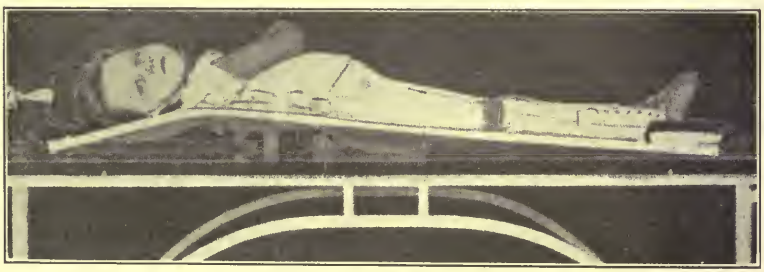

FIG. 715.— MODIFICATION OF FRAME SHOWN IN FIG. 714.
Traction applied to the head and limbs (Whitman).

body are obviated. Of the various means for accomplishing this purpose, placing the patient in horizontal fixation is the most effective.

Horizontal fixation apparatus has been devised by a number of surgeons (Lorenz,[3] Phelps,[4] Redard,[5] Whitman[6] and others.) Whitman,[6] whose experience in this connection is considerable, states that the bed

frame of Bradford and Lovett quite meets the requirements. This is a rectangular frame of gas pipe, a few inches longer and slightly wider than the patient's body. Covers of strong canvas are drawn tightly over the frame by means of corset lacings or straps on its under surface, leaving an interval beneath the buttocks for the use of the bedpan (Fig. 714). The frame may be bent to suit various postures and counter extension is easily employed in connection with it (Fig. 715). Apparatus of this sort is used in spondylitis in all stages in young children and, in older children, during the acute stage of the disease. When the acute symptoms have subsided, *ambulatory supports* may be employed; these consist of the *gypsum jacket and the steel brace.*

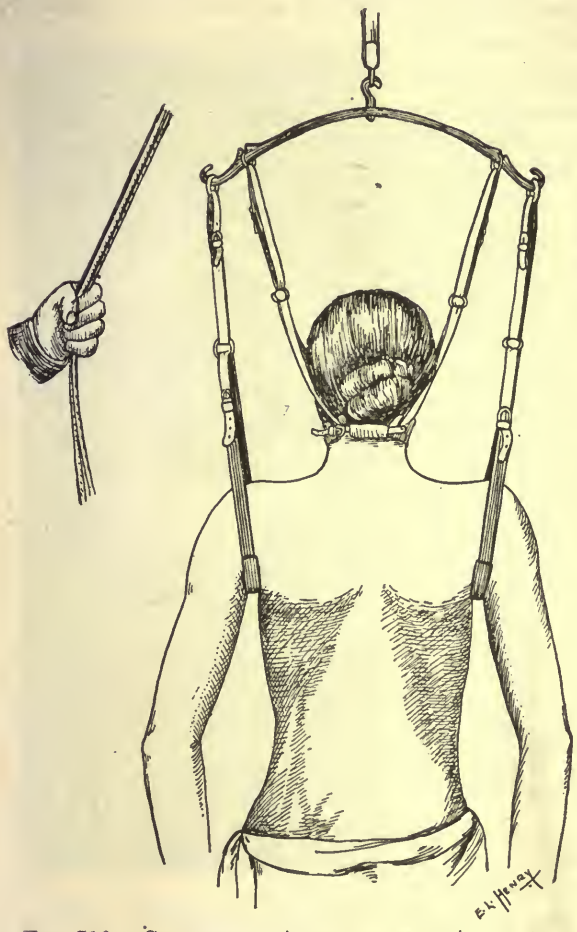

FIG. 716.—SUSPENSION APPARATUS FOR APPLYING GYPSUM.

The GYPSUM JACKET is applied so that it SUPPORTS THE SPINE BY HOLDING IT IN THE ERECT OR EXTENDED POSITION, THUS TRANSFERRING THE WEIGHT IN PART FROM THE DISEASED VERTEBRAL BODIES TO THE LATERAL AND POSTERIOR PORTION OF THE COLUMN (Whitman[6]). The efficacy of the jacket depends upon the accuracy of its adjustment and upon the leverage it exerts above and below the site of the lesion. A *tricot* shirt reaching to below the knees is first applied to the body. A

band of china silk or linen about three inches in width is placed beneath the shirt on the back and front. These bands, which Lorenz[3] calls "scratchers," may be manipulated beneath the jacket and keep the skin clean. The patient is then placed upon a stool and the halter of the suspension apparatus (Fig. 716) is adjusted, the arms are extended over the head, and the hands clasp the straps or rings. Sufficient tension is made upon the rope partially to suspend the body and to overcome the deformity as far as is practicable. The act of suspension should not cause pain (Sayre[7]). Before applying the gypsum, *pads of felt* or similar material are placed over the bony protuberances (anterior spines, sternum, etc.), and, finally, great care should be taken to pad thickly the surfaces immediately contiguous to the seat of the disease. In the female, the breasts (when present) are also protected. In very thin children, a pad is placed over the abdomen to allow for physiological increase in size in this situation. The *gypsum bandage,* which should consist of wide meshed crinoline (not gauze), into the meshes of which gypsum has been incorporated (by hand), is now molded in place. For the purpose, one person sits behind the patient and one in front, while the third may hold the guy ropes and control the tension and the position of the body. When the jacket is nearly firm, it is trimmed. As a rule, the apparatus should reach from the upper margin of the sternum to the pubes in front; behind, from about the middle of the scapula to the gluteal fold; laterally, it should be cut away sufficiently to prevent chafing of the axilla above and below, on either side of the pubes, and an oval section

FIG. 717.—THE "CALOT JACKET," SHOWING THE GYPSUM APPLIED TO THE NECK AND SHOULDERS (Whitman).

is excised to allow of comfortable flexion of the thighs. After the jacket is applied, the patient is kept in the recumbent position until the gypsum is quite hard (at least half an hour). The skirt of the shirt is then folded over the jacket and sewed to the protruding buried portion.

If the lesion is above the tenth thoracic vertebra, the gypsum is carried about the neck and in front of the shoulders (Fig. 717). In many instances, and always in disease of the cervical region, support of the head is necessary. For the purpose, the head may be included in the gypsum dressing or a so-called "jury mast" may be employed. This consists of a strong steel brace, the bottom of which is incorporated into the jacket below the seat of the disease (Fig. 718).

FIG. 718.—THE "JURY MAST" TOGETHER WITH THE ORDINARY GYPSUM JACKET (Whitman).

The essentials of the *Calot jacket* (Fig. 717) are fixation of the neck and shoulders as well as the pelvis, and direct pressure over the kyphos, the front of the jacket being cut away so that the trunk may be forced forward, thus straightening the spine as a whole and, to a certain degree, correcting the deformity at the seat of the lesion. In applying the apparatus, the patient is suspended as stated. If the head is to be included, a special sling is used. The molding of the gypsum is also performed as stated. After the front of the chest and abdomen are covered with a one inch layer of cotton batting, the arms are supported at right angles to the body. The gypsum is applied very thickly. When the jacket is sufficiently firm, the patient is

placed on his back and a small opening cut over the chest, through which the thoracic pad is removed; so that respiration may not be interfered with. The following day the front of the apparatus is cut away as shown in Figure 717. Another opening is made behind, exposing the seat of the disease; through this the skin is anointed and pads of cotton are forced into the opening to the point of tolerance, with the aim of pressing the trunk forward and of obliterating the kyphos. The pads are held *in situ* by a few turns of a gypsum bandage or by adhesive plaster. At intervals of several weeks, the pads

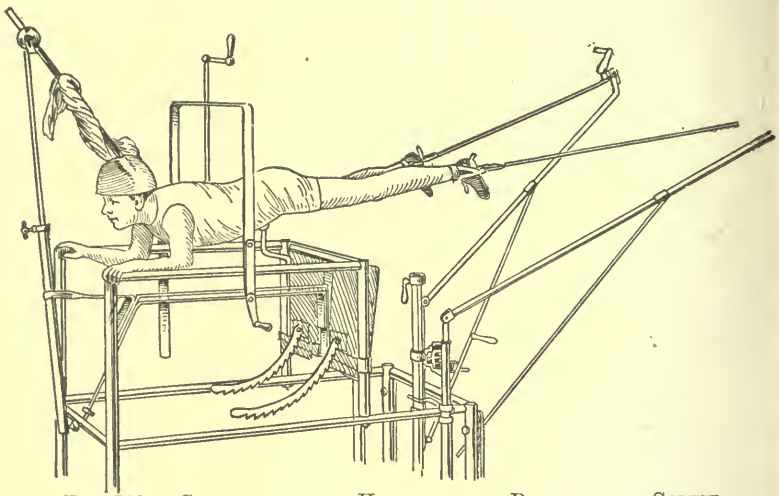

FIG. 719.—CORRECTION OF A KYPHOS WITH PELOTTE AND SCREW.

are changed, increasing, as far as possible, the pressure, with the view of still more obliterating the kyphos.

It is not infrequently necessary to *apply the gypsum apparatus with the patient in the recumbent position,* with or without traction. For the purpose a number of methods are *en vogue,* among which may be mentioned those of Taylor, Goldthwait and Metzger (see Whitman[6]), and the Hawley table. The Bradford-Lovett frame (Fig. 714) will be found very satisfactory for the purpose. The jacket may be worn for months (six) at a time; as a rule, it is necessary to replace it every two months.

In Europe, *Gaugerle's*[8] *method of applying gypsum* is largely used. The patient is postured on a specially constructed apparatus (Fig. 719), which is fitted with a pelotte and screw. By means of the latter, the kyphos is gradually corrected (without narcosis) and the gypsum

apparatus applied. Gaugerle[8] reports ten cases of deformity with paraplegia in which he obtained favorable results.

When the stage of recovery is reached, a *gypsum corset* may be worn. This is simply a removable gypsum jacket, which allows of intermittant use under the guidance of the surgeon.

Steel braces are the outcome of an effort to replace the gypsum jacket with "something different." Much ingenuity, and more literary and vocal efforts, have been expended in connection with their construction. They are not considered as taking the place of the gypsum apparatus in any regard. However, the one which has maintained a place in the textbooks for many years is that of Taylor (see Whitman,[6] who depicts several others) which is shown in Figure 720.

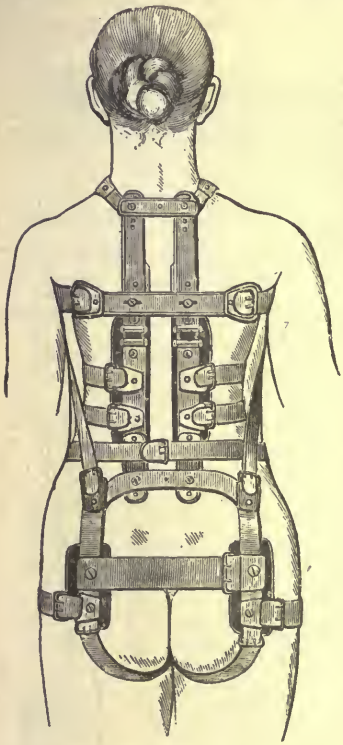

FIG. 720.— THE TAYLOR BACK BRACE (Whitman).

The *operative fixation of diseased vertebrae,* with the view of obviating the occurrence of kyphos, was first attempted by Lange[1] (1899), who fastened the contiguous spinous processes to each other by means of paraffin oxycyanid silk. Bradford[10] used bone plates in a similar manner. Hibbs[11] attempted to ankylose the vertebral arches by chiseling through the upper half of the spinous process and turning the fragment down, so that its point rested in a previously prepared groove of the next lower process. Bayer[12] regards the latter method as more desirable than the use of wires and other pliable materials. The technic of Albee[13] seems to overcome the objections raised in connection with the others. It is performed as follows: An incision is made, starting well above the lesion and curving to one side of the median line and carried back to the middle, well below the diseased area, thus forming a crescentic skin flap. Having dissected up the skin flap, the tops of the spinous processes and the supraspinous ligaments are exposed. The latter are split over the tops of the spinous processes, dividing them into equal halves; the interspinous ligaments are also

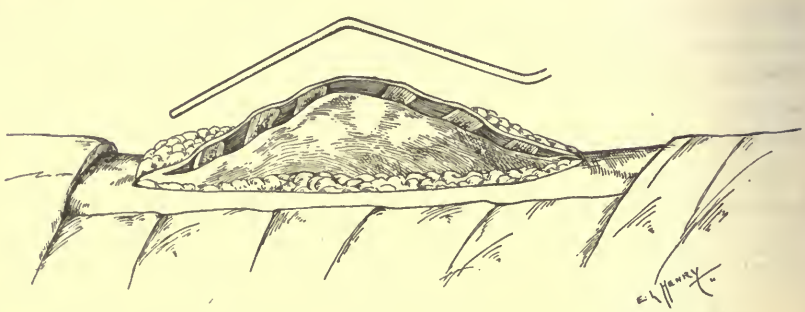

FIG. 721.— THE FLEXIBLE PROBE BENT TO CONFORM TO THE KYPHOS.

To be used as a pattern in removing the curved graft from the anterolateral aspect of the tibia (Albee).

split, care being taken not to damage additional muscles and ligamentous attachments to those processes. Then, with a thin, sharp osteotome, the spinous processes are split (vertically) to a depth of from one third to two thirds of an inch. One half of each process

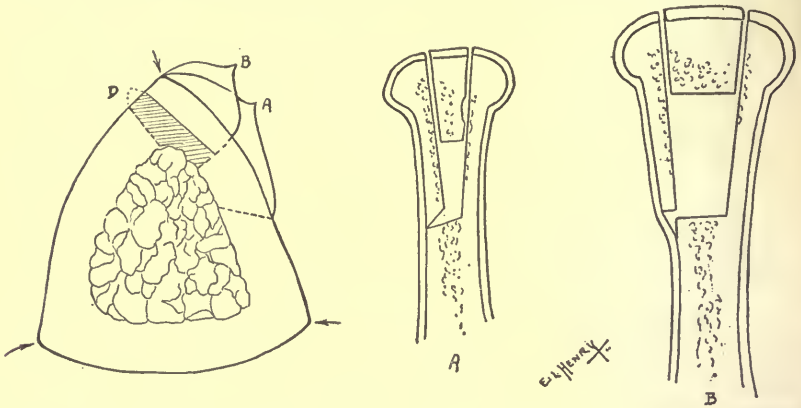

FIG. 722.— DRAWING OF CROSS SECTION OF TIBIA AND TIBIAL GRAFTS IN PLACE.

A is spinal graft for an early case that has not become kyphotic. B is cross section of graft which, on account of the large size of the kyphosis, is bent over it. D represents the multiple saw cuts on the marrow side.

A illustrates a cross section of a spinous process split in half and fractured at its base. The deep, thin graft in cross section has been removed from the crest of the tibia, having its periosteum attached to two sides. The side in contact with the unbroken half of the spinous process is the saw cut or the medullary surface of the graft.

B illustrates a cross section of a spinous process which has been split, and one half has been set over to produce a gap sufficient to receive a broad graft removed from the antero-internal surface of the tibia, having periosteum on one surface only; the medullary surface of the graft lies nearest the base of the spinous process in the gap (redrawn from Albee's Bone Graft Surgery).

(always on the same side) is completely fractured at its base and moved over a distance varying in accord with the thickness of the graft which is to be implanted. The *length and shape of the required graft is determined by calipers and a flexible probe is applied to the gutter bed.* The shape of the graft is likely to be nearly that shown in Figure 721. Using the bent probe as a pattern, a graft conforming to it is cut from the tibia with the twin saws, as shown in connection with the surgery of bones. The graft may be partially sectioned at intervals in order to conform it to the kyphos. The graft is so placed that the narrow, or saw cut, surface will be in contact with the side of the gutter formed by the unfractured halves of the spinous processes, and its periosteal surface makes contact with the opposite side of the gutter containing the fractured halves of the spines. The graft is held in place by several deeply placed sutures. The skin wound is closed with silkworm gut. Figure 722 shows the outline of a small and a large tibial graft; A shows a cross section of a spinous process with a small graft and B shows a process with a large graft embedded. A Röntgenogram of a graft embedded in the process of the cervical vertebra is shown in Figure 723. The behavior of the graft and its influence on genesis of bone in this situation are similar to those taken up in connection with the discussion of the problem as a whole (Part IV, chap. vi). The advantage claimed for the method is that ankylosis of the healed process takes place with less deformity than obtains

FIG. 723.—RÖNTGENOGRAM OF BONE GRAFT IN CERVICAL REGION (from Albee's Bone Graft Surgery).

when the spine is immobilized with mechanical appliances. Albee[13] advises that, *following the operation,* the patient be kept on a fracture bed for from five to six weeks, with no more restraint than that afforded by pinning about the thorax a towel, to which four strips of broad muslin bandage are attached. The restraining bandage strips are so placed as to prevent the patient from sitting up or rolling from side to side, and are usually necessary only with children. When the spine presents a marked kyphosis, it is necessary to apply thick pads on either side of the spine before placing the patient on his back; when the deformity is severe, the lateral position is best used. Active work is not permitted until the end of about six months. During this time it is not, as a rule, necessary for the patient to wear any apparatus; however, in severe cases there is no objection to the employment of a gypsum corset for a few months.

As already stated, spondylitis is mostly tuberculous in character. The treatment when the disease is due to other causes does not differ from that described above. It is probable that no other form of the disease is likely to be as prolonged as the tuberculous.

Distorsio articularis sacro-iliacae gives rise to radiating pain, which may be mistaken for that due to spondylitis. In many cases the pain involves the zone of distribution of the *great sciatic nerve* (Goldthwait[14]). In some of the cases the pain is relieved by a properly fitting support, although in not a few instances nothing seems to afford relief. No doubt the inaccessibility of the joint, which makes this a difficult problem, together with the uncertainty of the diagnosis, has acted as a deterrent in this connection. Lovett and Reynolds[15] consider that pain in the sacro-iliac region is due (in some instances) to faulty carriage of the body, the result of gynecological conditions, and advise that these be remedied before the case is condemned to hopelessness.

A special form of spondylitis is described by Lange[1] under the term *chronic traumatic spondylitis.* The disease occurs in adults following a severe injury. He likens the condition to that occurring (after injury) in the knee and ankle joints, in which the lesion is restricted to pathological processes in the periosteum and ligaments. In a certain number of cases, the disease is an already existing mild *spondylitis deformans* (whatever that may mean), which is simply accentuated by the injury; it usually occurs in elderly persons.

Bechterew and Strümpel[16] describe a condition of the spinal column which they designate as *chronic vertebral ankylosis.* It appears in

the literature as *spondylitis, and spondylarthritis ankylopoetica, and spondylose rhizomélique* (Pierre Marie[17]). *Histologically,* the disease does not differ from the picture presented in connection with chronic inflammatory processes involving bones and joints in general. That is, we probably are not confronted with a clinical, nor, indeed, a pathological entity, but with the result of syphilis, rheumatism, gonorrhea, influenza, etc. If this is true, then the discussion entered into above, as to the desirability of recognizing the character of the causative factor in spondylitis as a whole, is worth heeding.

The condition is attended with *pain, interference with function, and deformity;* the last often going on to kyphosis, especially in old persons. The muscles of the back of course atrophy. It is not at all improbable that the person "bowed by the cares of a long arduous life" is often afflicted with chronic spondylitis which had its origin in nothing more emotional than the colonization of the gonococcus or the spirocheta pallida, and an early diagnosis and appropriate treatment might have arrested the progress of the disease.

For rachitic spine see Rachitis, page 1074.

For scoliosis see The Surgery of Deformities, Part VIII, chap. viii, p. 1370.

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CHAPTER IV

INJURIES OF THE SPINAL CORD

Concussion of the Spinal Cord.— The question as to whether there is such a thing as pure concussion of the spine has been made the subject of much discussion. A reiteration at this time of the various views in this connection does not serve a useful purpose. Frazier[1] summarizes the situation as follows: "When there is injury to the spinal column without a bony lesion but with subsequent alteration in the spinal cord, it were well to class such cases as *contusion* and to eliminate the term *concussion* as applied to the spinal cord altogether." On the other hand, there is a class of cases of injury of the spine, the pathological anatomy of which is not discoverable, that are described in the literature under the head of *"railway spine."* Irrespective of whether the condition is attended with minute hemorrhages or is the outcome of commotion of the spinal cord or not, *it certainly represents a clinical entity* and may be described as such.

Usually, the *clinical history of "railway spine"* is that the patient in a railroad accident is thrown violently forward out of his seat and against the back of the seat in front, or, in cases of rear end collisions, he is subjected to a violent impact along the entire spinal column. Aside from the objective contusions, etc., there is always severe fright, and this must not be overlooked or belittled. The patient may be able to get up and walk to a place of safety after the accident, but the disturbance to his nervous system is very apparent and, though objectively uninjured, he feels weak and dizzy, and at times evinces great "emotionalism," weeping or laughing — possibly, both together. Often, though helping to extricate his more unfortunate companions, he is greatly nauseated and vomits.

From this on there are four possibilities. (1) He may rapidly become perfectly normal and show no ill effects from his experience; (2) he may exhibit signs of organic involvement of the spinal cord, which symptoms may increase in severity as time goes on (traumatic spinal apoplexy); [manifestly, these two eventualities have nothing

to do with railway spine] (3) he may become the subject of traumatic *neurasthenia;* and (4) he may suffer from *traumatic hysteria.* The last two contingencies open a wide field, one which leads to a discussion far beyond the possibilities of this book. Suffice to say that the disturbances following the class of injury spoken of are real; that they are the outcome of a disturbed nerve function; and that they present a complex symptomatology which includes modification of motion and sensation and inactivity of the viscera of the exhaustive type.

Hysterical railway spine is a still *more confusing* problem than the so-called *neurasthenic type.* It is taken up *in extenso* by Babinsky and Froment[2] in connection with *war injuries.* Their work also furnishes a complete bibliography on the subject.

Hematomyelia.— Hematomyelia may be defined as a *hemorrhage* in the spinal cord following trauma to the spinal column, *with or without a lesion of the spinal column.* The mechanical explanation of its occurrence may be sought for in the fact that a shock is transmitted to an incompressible column of fluid contained in delicate elastic tubes which, particularly in the gray matter of the spinal cord, receive but meager support from the surrounding tissues.

The *effusion of blood in hematomyelia* varies in size from a small group of erythrocytes (microscopical) to a cavity filled with blood occupying over one half the transverse diameter of the cord. The perpendicular extent of the bloody effusion may be restricted to one segment or may invade several of them. As a rule, the damage to the gray matter is the greater (Allen[3]).

The *symptoms* of hematomyelia relate of course to impairment of the function of the part dominated by the injured area. These do not differ in any regard from those attendant upon other lesions, except perhaps in the fact that, IF AN APPRECIABLE PERIOD OF TIME ELAPSES BETWEEN INFLICTION OF THE TRAUMA AND THE ONSET OF THE SYMPTOMS, THE PRESENCE OF HEMORRHAGE MAY BE ASSUMED. The severity of the symptoms indicates the extent of the hemorrhage. A clinical working basis as to localization is presented farther on.

The *prognosis* in uncomplicated cases, as far as life is concerned, is good. The period of absorption of the effusion of blood is very variable (six to ten months). Destroyed nerve cells are not regenerated.

The *treatment* aims at complete rest until the acute symptoms sub-

side. The distorting effect of the incompletely or completely paralyzed muscles must be obviated. *Massage* is useful in all cases.

Contusion of the Spinal Cord.— The term contusion of the cord is applied to injuries in which there is no solution of continuity. Allen[3] has shown that small impacts, sufficient to cause complete motor or sensory paralysis, with initial loss of all reflex activity below the level of the injury, may not be inconsonant with complete recovery. As a rule, CONTUSION OF THE CORD IS THE MOST IMPORTANT FACTOR IN FRACTURE OR DISLOCATION OF THE VERTEBRAE; it may, however, occur without either of them.

Laceration of the Spinal Cord.— The cord may be lacerated by gunshot projectiles; or the laceration may be incidental to a stab wound, or to a fracture or dislocation of the vertebrae. In the last instance, the osseous displacement is greater than in contusion. The laceration of course varies in degree and is attended with a *clinical picture* in this proportion. In slight lacerations, the prognosis, as regards life, is not bad; when the cord is completely torn, a fatal outcome usually supervenes in a short time. In a general way, it may be said that *the higher the injury, the graver the prognosis.*

An *exact diagnosis* as to the degree of laceration is not always possible, as, especially in gunshot wounds, contusion and laceration coëxist. In gunshot wounds impact of the projectile may severely contuse the cord without perforating the dura. Particularly interesting experimental work has been done in this connection by Allen.[3]

The Symptomology of Complete and Partial Cord Lesions.— It may be said that the symptomology of cord lesions is dependent upon the extent of destruction of tissue, irrespective of the character of the causation, and therefore, for convenience, is discussed under one head. In studying this somewhat complex problem, it is desirable to bear in mind that the EARLIER A CLEAR CONCEPTION OF THE MOTOR AND SENSORY SYMPTOMS IS RECORDED, THE MORE READILY IS A CONCLUSION POSSIBLE AS TO THE ADVISABILITY OF MAKING OPERATIVE EFFORTS AT RELIEF.

The symptoms bearing on the question as to whether the lesion is a complete or an incomplete (or partial) one are formulated by Frazier[1] as follows:

COMPLETE	PARTIAL
Usually flaccidity of all muscle groups whose innervation comes from segments below the level of the lesion. Paralysis is complete.	A spastic condition with or without contractures of muscle groups whose nerve supply comes from segments below the level of the lesion. Paralysis may not be total.
Loss of reflexes whose arcs lie in segments below the level of the lesion.	Increase of reflexes whose arcs lie in segments below the level of the lesion. Presence of the Babinsky phenomenon.
Complete loss of control of bladder and rectum, which is demonstrated by either constant dribbling or by periodic evacuation without knowledge or volition.	A possible partial control of bladder and rectum, with at times knowledge on part of patient that evacuation is taking place.
Complete loss of all forms of sensation of a sharp segmental distribution.	Loss of sensation is not absolute, in that the patient often perceives strong stimuli of protopathic nature, and is at times able to detect changes in position.
The symptom picture is stationary with a tendency toward trophic changes for the worse.	The symptom picture is not necessarily stationary, and a gradual improvement may be noted.

It remains to consider the *symptoms which indicate the level of the lesion.*

In lesions of the fourth cervical region the motor symptoms are those of a complete quadriplegia. As the main origin of the phrenic nerve lies in the fourth cervical segment, there will be total paralysis of the diaphragm, and breathing will be costal in type. The *sensory symptoms* will be complete loss of all forms of sensation up to and including the skin area supplied by the fourth cervical vertebra (Fig. 724). In transverse lesions in the cervical region it must be remembered that the fibers from the midbrain to the *celiospinal center* in the eighth cervical and the first thoracic segments are sectioned and that, although the celiospinal reflex shares to a certain extent the automatic action of the other autonomic mechanisms, in the cases of high cervical lesions we are likely to have *bilateral contracted pupils.*

In lesions of the fifth cervical segment the symptoms are practically the same as when the fourth segment is involved, with the exception

that the major part of the phrenic nerve escapes, and therefore the *diaphragm is not paralyzed.*

In lesions involving the sixth cervical segment, the upper root of the brachial plexus escapes, and consequently partial preservation of the power in the shoulder girdle obtains. In other respects the clinical picture is the same, except that *tactile sense is preserved on the upper and outer surface of both arms.*

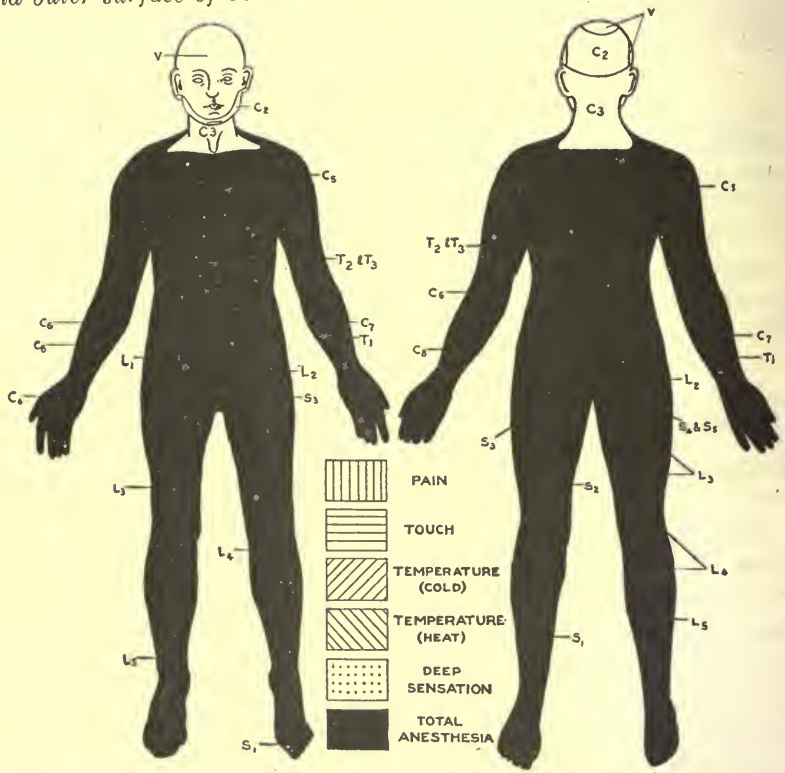

FIG. 724.—SENSORY DISTURBANCES FOLLOWING AN INJURY OF THE FOURTH CERVICAL SEGMENT (Frazier).

In lesions involving the seventh cervical segment, the *biceps tendon reflex is preserved.* This is at times exaggerated. The patient is very likely to assume the position shown in Figure 725. The sensory disturbances are peculiarly significant and are shown in Figure 726. In cases of injury it is necessary to bear in mind that effusion of blood may provoke the appearance of symptoms emanating from the segment above.

In lesions involving the ninth thoracic segment there is of course no paralysis of the upper extremities. However, there is complete paralysis of the lower extremities which, as in injuries of higher levels, is either *flaccid* or *spastic*. The *sensory* symptoms are as mentioned above (Fig. 727).

FIG. 725.— POSITION OF THE UPPER EXTREMITIES IN A COMPLETE TRANSVERSE LESION OF THE CORD AT THE LEVEL OF THE SEVENTH CERVICAL SEGMENT (Frazier).

In the lesions already recorded, the bladder and rectum symptoms are very similar. As the pathways of sensory conduction from bladder and rectum are interrupted, the patient does not feel distention of either. At first (the reflex activity being set aside) there is a tendency to the incontinence of retained material after a certain amount of accumulation of excretion has occurred.

In lesions of the second and third lumbar segments the *motor picture* is one of complete paraplegia, which is likely to be of the flaccid type. The *sensory disturbance* is limited to a line corresponding to Poupart's ligament (Fig. 728). As the centers of micturition and defecation are located in the first and second lumbar and the second and third sacral segments, the disturbances of urination and defecation are very serious.

The *Brown-Séquard syndrome* is present in lesions involving more or less definitely one half of the cord. The symptoms presented by

such a lesion in the fifth or sixth thoracic segment would be as follows: Complete paralysis of the right lower extremity — most likely of the flaccid type; some diminution or loss of tactile sensation on the right, extending up on the trunk to the cutaneous distribution of the fifth thoracic nerves (this tactile anesthesia will be neither profound nor permanent); a loss of pressure, position, and osseous sense on the right below the level of the lesion; loss of thermal and pain

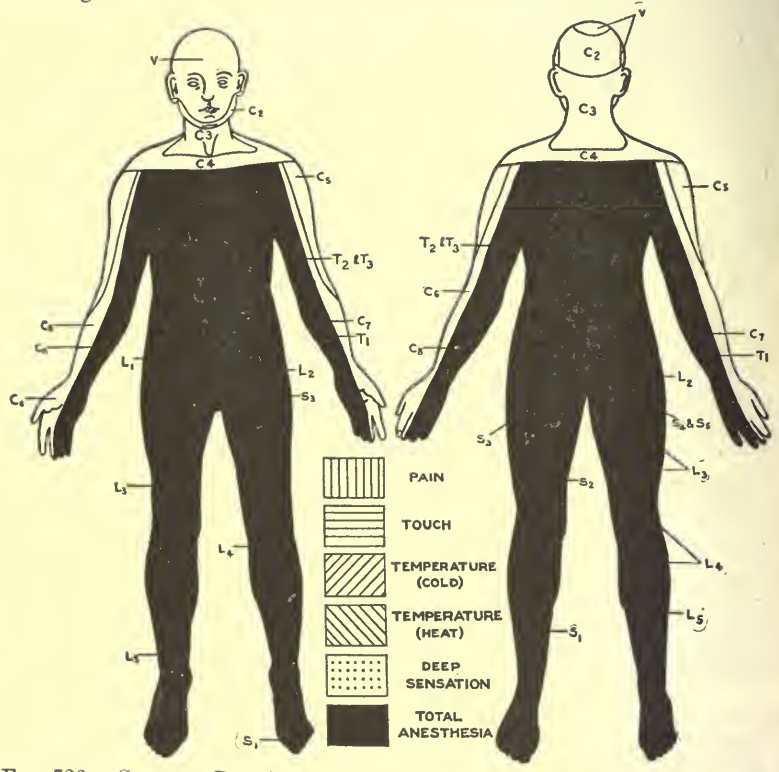

FIG. 726.— SENSORY DISTURBANCES FOLLOWING A LESION OF THE SEVENTH CERVICAL SEGMENT (Frazier).

sense on the left up to a line somewhat lower than the sensory involvement on the opposite side. In addition to this, there are certain sensory disturbances forming a zone of variable width between the normally innervated skin above and the anesthetic areas below (Fig. 729).

Lesions of the Cauda Equina.— In lesions below the second lumbar vertebra the substance of the spinal cord is not involved, as this structure ends at about the disc between the first and second lumbar ver-

tebrae. However, here the spinal nerves form a great collection, encircling the conus and running down the canal to their points of exit. It follows, then, that a lesion in this situation, in accord with its exact location, may involve all or only a part of these conducting elements, and that the motor and sensory symptoms would correspond to the seat of the involvement. A lesion located between the second and third lumbar vertebrae is characterized by involvement of the

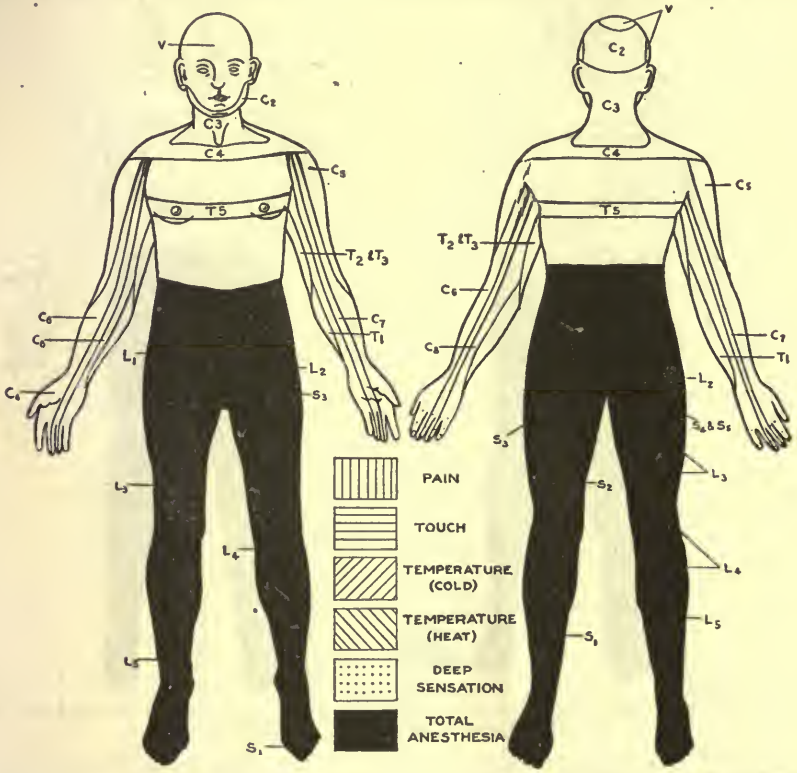

FIG. 727.— SENSORY DISTURBANCES FOLLOWING AN INJURY OF THE NINTH THORACIC SEGMENT (Frazier).

entire lumbosacral plexus and by paralysis of the bladder and rectum. *Sensation* is lost below Poupart's ligament. If the lesion is below the fifth lumbar nerve, the motor disturbances are restricted to the lower legs and feet, and sensation on the dorsum and sole of the foot and a strip on the back of the leg is impaired or lost.

The *treatment of lesions of the spinal cord,* other than the general care of the patient, already discussed in connection with fractures

(Part IV, chap. v), relates to the question of operative measures of relief. *In cases of injury,* as already stated, this is not easily decided. In arriving at a conclusion, the surgeon must make a careful study of the sequence of symptoms as they develop from the time the injury

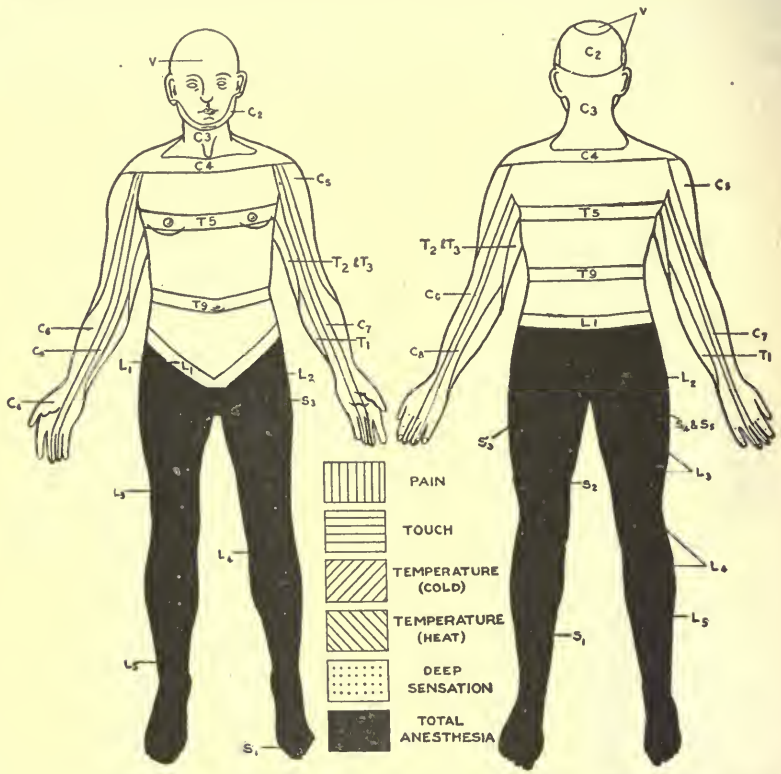

FIG. 728.— SENSORY DISTURBANCES FOLLOWING AN INJURY OF THE SECOND AND THIRD LUMBAR SEGMENTS (Frazier).

was received and interpret them as balanced against the actual physical possibilities connected with his available measures of relief which, after all, consist simply in the removal of compressing agents, i. e., foreign body, bone, blood and exudate. If, therefore, an intensive study of the case indicates that the attempt to remove the offending agent is justified, he may, with a clear conscience, proceed to operate. It is here reiterated, that surgical repair of divided or destroyed nerve centers is not followed by regeneration. For the technic of the exposure of the spinal canal see *Laminectomy* (p. 1472).

Stab and Gunshot Wounds of the Spine and Contents of the Spinal Canal.— The *distinctive clinical factor* in stab and gunshot wounds of the spine *relates to injury of the cord and its membranes,* of which of course the former is the more important. Therefore, the subject is taken up at this time.

Stab Wounds.— Stab wounds are inflicted by sharp or pointed in-

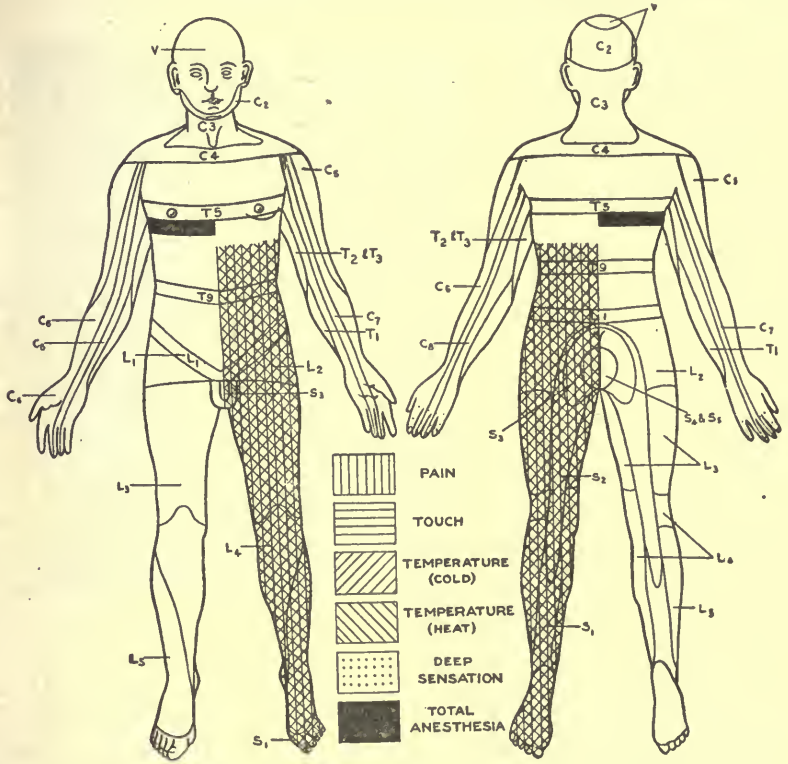

Fig. 729.— Brown-Séquard Syndrome in Lesion of the Sixth Thoracic Segment.

On both sides below the cutaneous distribution of T_5 there may be ill defined areas of tactile anesthesia (Frazier).

struments, such as a sword or a knife. Of 93 cases collected by Petrén,[4] 43 were in the cervical and 50 in the thoracic region. The osseous injury does not present any peculiar surgical problem; the importance of the lesion relates to contusion and puncture of the membranes and to involvement of the cord, which may be a complete section. As a rule, the instrument enters the canal between the arches of the verte-

brac; however, it occasionally happens that an arch or a spinous process is fractured. Not infrequently, a portion of the instrument is left in the vertebra, sometimes partially in the canal.

Extradural hemorrhage is usually insignificant. Occassionally, there is a *cerebrospinal fistula* with free escape of the fluid, which presents great danger of infection. The fact that cerebrospinal fluid does not escape from the wound does not exclude the possibility of penetration of the dura. On the other hand, the cord may be severely contused and the dura remain intact.

The symptoms, when the canal is penetrated and the cord injured, are those of *immediate paralysis.* These vary in accord with the location of the trauma and do not differ from those following other injuries. After a short time the Brown-Séquard syndrome is very likely to make its appearance. This is very easy to understand from the nature of the injury (Rauzier and Rimbaud[5]).

The *treatment of stab wounds* of the spinal contents must take into consideration the wound, the presence of a foreign body, a cerebrospinal fistula and injury to the cord. *The majority of these wounds are not infected and must simply be kept clean. Probing is not permissible;* a Röntgenogram must be taken at once to determine the presence of a foreign body and its location.

Operative measures are indicated (see Laminectomy, p. 1472) when the Röntgenogram reveals the presence of a portion of the instrument in contact with the membranes or the cord. In the absence of this guide, evidence of pressure on the cord may be regarded as an indication to operate. The general care of the patient is important (see Fractures, etc., of the Spine, Part IV, chap. v).

Gunshot Wounds.— Gunshot wounds of the spine and spinal cord are among the most serious of war injuries. They occur at every level, although they are most frequent in the thoracic region. Statistical analyses in this connection are furnished by Exner,[6] Prewitt,[7] and Haynes and Moorhead.[8]

The *bone lesions* depend upon the portion of the vertebra involved and upon the character of the projectile. The arches and spinous processes are more often fractured than the bodies.

The *cord lesions* are of course the important factors in this class of injury. The cord may be *severely lacerated* or even completely severed by direct contact with the bullet or by pieces of bone driven into it by the projectile; it may be compressed by displaced fragments of bone, by the bullet protruding into the canal, by a subdural hemor-

rhage, or by adhesions or serous exudates; it may be *contused* by the bullet and splinters of bone; and, finally, the cord may undergo structural changes from the effects of *concussion or commotion* caused by the impact of a projectile striking against some portion of the vertebral column and bounding back to become lodged in the soft parts; or the *sudden atmospheric changes* caused by the explosion of shells in the vicinity of the patient may result in changes in the spinal cord. Jumentie[9] reports a case of indirect injury of the cord at some length; its history is repeated by Frazier.[1]

FIG. 730.— X-RAY SHOWING BULLET BETWEEN SECOND AND THIRD CERVICAL VERTEBRÆ, IN A VERTICAL PLANE CORRESPONDING TO THE REGION OF THE SPINAL CORD (after Perthes).

The position of the bullet corresponds to the level of the cord lesion (Frazier).

Laceration and contusion of the cord by a projectile does not differ from that produced by other agents, except that they are likely to be very severe.

Compression of the cord by a projectile produces, first, irritation, and later, pressure. If the pressure is not promptly removed, edema, myelitis, and, finally, necrosis and other grave degenerative changes take place. *In no case is it permissible to allow the bullet to remain in situ;* sooner or later, it will give rise to inflammatory exudates, adhesions, and thickening of the dura, which eventually extend to the cord. Bullets give rise to extensive hematomyelia.

In some cases the disturbances caused by the bullet are attended with adhesions of the leptomeninges and collections of fluid, presenting a picture of *serous meningitis.* The collection of fluid causes pressure symptoms, which demand early intervention.

Concussion of the cord in connection with bullet wounds of the spine, in which there is no bone lesion or only an insignficant one, has been

carefully studied during the "World War." In most of these cases, the projectile has struck the vertebral column and rebounded to become embedded in the soft tissue, or in an organ more or less remote from the spine, but the impact of the bullet is so great that the vibratory force is transmitted to the cord, resulting in *medullary lesions varying from slight degrees of contusion to complete disintegration.* Many theories have been advanced as to the cause of these changes. These comprise the possibility of a momentary displacement of the vertebrae (temporary subluxation) and the belief that the cord is caused to oscillate within the canal (Fickler[10]). Frazier[1] regards the latter as a reasonable explanation. Claude and L'Hermite[11] suggest that the shock transmitted to the cerebrospinal fluid and the temporary increase in pressure bring about an ischemia sufficient to cause insulatory necrosis of the marginal vessels. This theory would seem to be supported by a case reported by Latarjet[12] and published in English by Frazier.[1] Mussen, also quoted by Frazier,[1] reports the histological findings in a case of this sort. The lesions consist in hematomyelia, edema, myelomalacia, disseminating myelitis, acute traumatic necrosis, hematorrachis, and arachnitis serofibrosa spinalis (see also Oppenheim[13]). It also not infrequently happens that these cases develop changes in the meninges and abnormal collections of fluids. The circumscribed form of serous meningitis has been studied by Marburg[14] and Rumpf.[15]

The *symptom complex* following gunshot wounds of the spine and cord does not differ materially from that attendant upon other injuries. *Pain,* the result of irritation of the spinal roots, is likely to be severe. For the purpose of localizing the cord injury see page 1450.

In the *diagnosis,* the *Röntgenogram* is of great assistance, especially in determining the question of whether the cord is invaded by the projectile or by splinters of the bone.

The *operative treatment* is determined by a judicious consideration of all the factors. *In indirect injuries, early operation is contraindicated.* If evidence of increasing pressure (from blood or exudate) develops, the canal may be opened. In *direct injuries,* the propriety of laminectomy is established (Frazier[1]); this view is strongly supported by Oppenheim.[13] The operation should be performed as soon as the shock of the injury subsides (see Laminectomy, p. 1472). For additional literature see bibliography of Frazier.[1]

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CHAPTER V

SPINAL TUMORS

Although tumors of the spinal cord, of the meninges and of the vertebral column are still regarded as rare, it is not improbable that inadequate diagnostic measures are to a considerable degree responsible for this conception. However, the progress in this connection since Horsley reported the first removal of a spinal tumor (1887) is very encouraging. This is to no little extent due to the researches of Déjerine,[1] Sherrington,[2] Head,[3] and others. Frazier,[4] in a search of the literature, found 330 cases of tumors in which operations had been performed. Of these, 152 were meningeal, 58 vertebral, 36 intramedullary, 30 caudal and 54 in which the origin was not stated.

The *etiology of tumors of the spinal cord* may be regarded as little understood, as is the case in tumors in general (see Tumors, Part VI).

As to *frequency of location,* Frazier[4] presents a series of statistics. In one table (Schlesinger's[5]) the interesting fact is recorded that of 135 tumors of the spine 91 were vertebral in origin.

Regarding the *character of the tumors involving the spine,* almost all classes of new growths are represented in the 330 cases collected by Frazier.[4] Excluding sarcoma, those belonging to the fibroblastoma class are the most frequently found; strange to say, only one was a gumma. Metastatic carcinoma and sarcoma of course make up a not inconsiderable proportion of the tumors in this situation. The former frequently occurs in connection with mammary carcinoma. A notion of the appearance of a spinal tumor *in situ* may be gathered from figure 731.

The *symptomatology* of spinal tumors, whether extramedullary or intramedullary, is sooner or later manifested through involvement of the spinal nerve roots and their respective segments in the spinal cord. The damage they inflict upon the tissues of the cord takes at first the form of *irritation,* then of *compression, encroachment,* or *destruction,* depending upon the location and character of the growth.

Frazier[4] describes *three established cycles in the life history of the spinal tumor.* The *first, or root, cycle* is the longest, and the symptoms, usually unilateral in the early stages, are those of irritation, and,

later, of compression. These are paresthesia, pain, hyperesthesia, and anesthesia in the areas innervated by the affected posterior roots; also

FIG. 731.— AN ENDOTHELIOMA DRAWN IN SITU, BEFORE ITS REMOVAL FROM THE SPINAL CORD AT THE LEVEL OF THE THIRD CERVICAL VERTEBRA (University Hospital).

The tumor appeared to take its origin from the dura in the lateral aspect of the spinal canal (Frazier).

tremors, muscular spasm and spasticities followed by muscular atrophy and paresis in the distribution of the involved anterior roots. As tumors are usually situated on the posterior surface, the motor symp-

toms are less frequent than the sensory. The *second cycle* is that of the *Brown-Séquard syndrome* (Fig. 729). This cycle is almost constant, unless the tumor is intramedullary. Oppenheim[6] has known only one case of extramedullary tumor in which the third stage was not preceded by the Brown-Séquard syndrome. The *third cycle* is characterized by weakness, and, later, by paralysis of the bladder and rectum, at first exaggerated, and, finally, by abolition of all reflexes whose arcs are involved and by vasomotor and trophic disturbances. Of the symptoms, constriction of girdle sensations are among the most distressing and are likely to mislead the practitioner into directing efforts at relief to the parts to which the pain is referred, such as operative attack upon the stomach, the gallbladder, the kidney, and the urinary bladder. The motor and sensory symptoms indicative of the location of the tumor are taken up on page 1450. However, the determination of the level at which a tumor is located is a problem of considerable magnitude; one which requires patient study and careful, thorough, frequently repeated examinations, by which the sometimes very gradual progress of the lesion may be recognized. Aside from the usual so-called neurological examination, that of the spinal fluid (lumbar puncture, p. 1414) must not be omitted, especially with regard to lues.

The *treatment* of spinal tumors is restricted to operative efforts at relief. Frazier[4] says "Excluding such lesions as tabes dorsalis or poliomyelitis, conditions usually clearly recognized and presenting no surgical indications, there remains a group of lesions of which tumors constitute a large proportion and in which the signs and symptoms point to a distinctly localized lesion, that is either driving the patient to distraction through pain, or destroying the cord by pressure." There are of course a number of cases in which the precise nature of the lesion, before operation, is impossible of recognition; nevertheless, *exploratory operation should be performed in all doubtful cases.* Exploratory laminectomy is of itself free from peculiar dangers; the time has passed when fear of opening and exploring the dural sac should be harbored. The earlier the operation, the better the outlook in every conceivable way.

The indications for operation may be summarized in a few words as being the recognition and localization of a lesion, the symptom complex of which indicates a new growth or even, with certain restrictions, a chronic inflammatory lesion in a patient whose condition is not prohibitive of any operative undertaking. The location of the

lesion in the upper segments is not a contraindication, as tumors have been successively removed from the level of the axis vertebra. Nor is it justifiable to withhold operative measures because the lesion is believed to be intramedullary, as this belief is not always correct and also because even an intramedullary growth may be favorably influenced by removal or by nerve decompression. The first intramedullary growth was removed by von Eiselsberg[7] (1907), since when 36 cases have appeared in the literature (Frazier[4]).

In *secondary* (metastatic) tumors, the decompressive operation is of value in a palliative sense, especially with regard to pain. The posterior roots may be divided at the same time. Laminectomy is described in chapter vii. (For additional literature see bibliography of Frazier[4]).

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CHAPTER VI

SURGERY OF THE SPINAL ROOTS

The surgery of the spinal roots is still in its initial stage of development. However, the student should realize that the subject is full of possibilities, especially with regard to the relief of spasticity, which, when attained, is among the most gratifying in the practice of surgery. Following recognition of the importance of the posterior roots in the conduction of *pain*, the question of directly attacking them came under consideration, with the result that Abbe[1] (1888) and Bennett[2] in the same year divided these structures. Abbe's case was free from pain for two years. Bennett's case died from cerebral hemorrhage on the twelfth day following the operation. In 1908, Förster[3] suggested that resection of the posterior roots be applied in severe forms of *spasticity* and described the technic Tietze[4] had worked out on the cadaver. Shortly after this Förster[5] applied the method for the relief of gastric crises.

Rhizotomy for the relief of pain has been employed in neuralgia of the upper and lower extremities, intercostal neuralgia, intractable pain in amputation stumps, neuritis of traumatic and luetic origin, pain due to herpes zoster and to pressure from inoperative sarcoma and carcinoma. The results of the operation for the relief of pain have not fulfilled the expectations of the inceptors of the operation; the procedure has been found to be much more effective in the treatment of spasticity and visceral crises. This does not mean that pain has not been relieved; on the contrary, a number of cases are reported in which the patient was entirely relieved; but it does mean that the exact source of irritation was not located in all the cases, and that this must be established before operative measures of attack may be expected to afford relief. The reports of cases include operations by Abbe,[1] Chipault,[6] Jones,[7] Mayo,[8] Horsley,[9] Giordani[10] and Hildebrand.[11] Of the cases operated upon for pain in amputation stumps, those analyzed by Knapp,[12] Bennett,[2] and Kilvington[13] may be studied with advantage.

Rhizotomy for the relief of spasticity has been practiced most often in connection with the various spastic affections of the central nervous system, spastic paraplegia and diplegia of congenital origin (Little's disease). Frazier[14] has collected seventy cases of Little's disease in which rhizotomy has been performed; two of which were practically cured, fifty-six were improved, three were unchanged, and nine died. In estimating these results, the factors adduced by Déjerine[15] may well be taken into account. Déjerine quite properly says that "Little's disease is not really a disease, but rather a syndrome and this makes an appropriate method of treatment very difficult."

In the acquired forms of infantile spastic conditions, section of the posterior nerve roots is not as effective as in the congenital types. In Frazier's[14] collection of thirty-five cases, twenty-five were markedly improved, no change occurred in six, and four died.

In hereditary and familial spastic paralysis, in *multiple or disseminated sclerosis,* and in *myelitis,* division of the posterior roots is only palliative.

With regard to the *selection of roots* to be divided, Frazier[14] *cuts two out of every three possible sources of sensory stimulation,* or, to put it in a different way, two of the three roots from which a given group of muscles derives its sensory supply.

After division of the sensory roots there is always more or less pain until the proximal stump of the root has completely degenerated; this lasts for four or five weeks. The spasticity will be varyingly under arrest from the beginning, the involuntary flexion of the limbs disappears, and voluntary movements, to a certain degree, gradually return. The preservation of the reflex defense indicates failure. The essential features of *the after treatment* are massage, faradization, passive movements, and mechanical and educational exercises.

For the relief of gastric crises in tabes dorsalis, rhizotomy has been resorted to with varying success. Of 73 cases, the records show that 14 were cured, 31 were improved, 5 were unimproved, and 16 died; of the last mentioned, 10 were operative deaths, and of the remaining 6, 3 were improved up to death. The methods of surgical attack upon the cord include intradural rhizotomy, extradural rhizotomy, extraction of the intercostal nerves, chordotomy, extradural gangliectomy, and ligature of both anterior and posterior spinal roots. Of these, the first seems to hold out the greatest hope of relief. A fair appraisal of intradural rhizotomy may be expressed in these terms:

two out of ten cases are cured, four are improved, three are unimproved, and one dies (Frazier[14]).

Intradural anastomosis for the relief of paralysis holds out a fascinating possibility. There are as yet only two cases on record in which the results are reported, one by Kilvington and Bird,[16] and one by

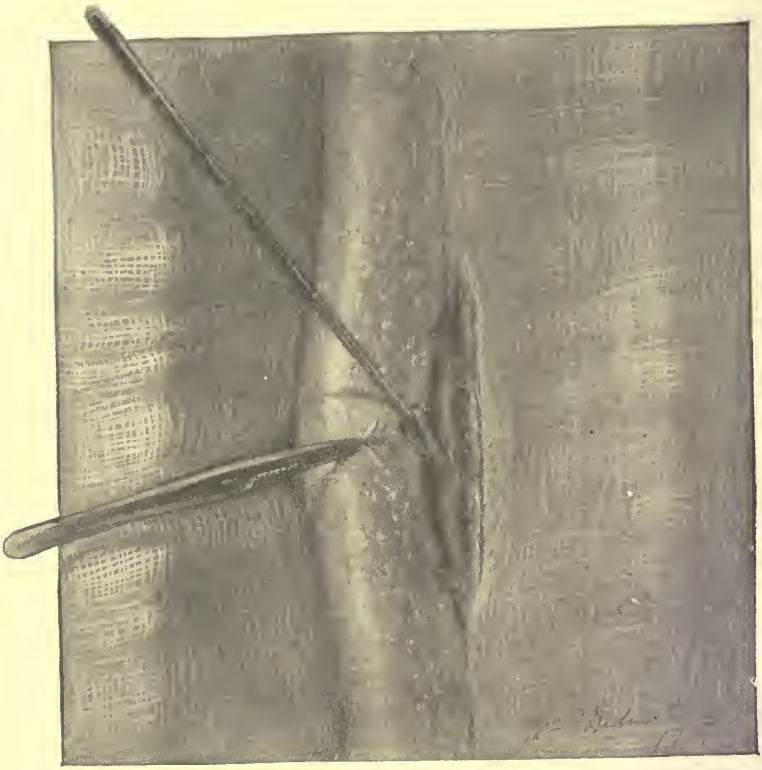

FIG. 732.— EXTRADURAL RHIZOTOMY.

The dura is grasped with mosquito forceps, with which the cord is rotated and drawn to the opposite side. With a blunt hook the posterior root is separated from the anterior root. The extradural rhizotomy requires a wider exposure and the separation of the roots is more difficult because of the prolongation of the dural sheath (Frazier).

Frazier.[17] The technic of the procedure includes the identification of the innervation of the paralyzed muscles and the proper selection of the substitute fibers — a problem of considerable difficulty. Frazier[14] describes in great detail the technic employed by himself (see bibliography).

Technic of Rhizotomy.— Rhizotomy implies cutting of the rhizome, or root, and, as a matter of fact, more than complete division is unnecessary; after the section is made, there can be no physiological union with return of function.

The *extradural method* (Figure 732) is supported on the ground that there is less risk, less danger of infection, and less danger of

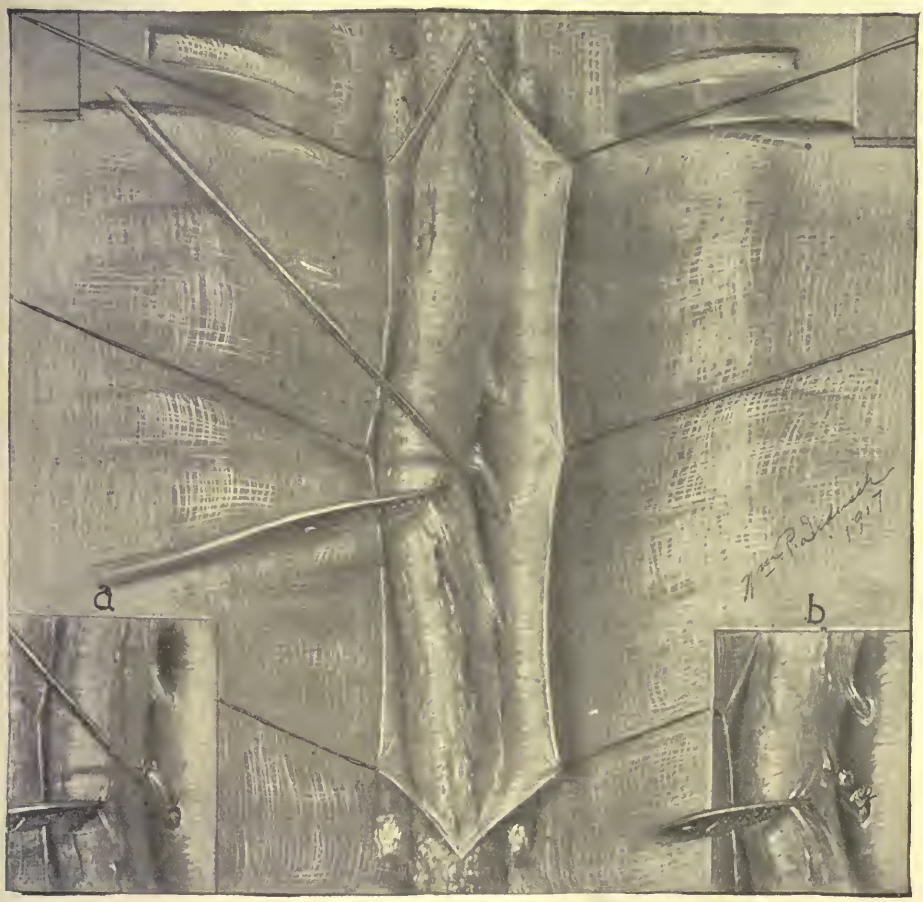

FIG. 733.— INTRADURAL RHIZOTOMY.

In the central figure the denticulate ligament is grasped with mosquito forceps, moderate traction on which rotates the cord and exposes the roots at their point of emergence from the cord and exit from the dural sac. With a small blunt hook the posterior root is separated from the anterior. *a*, A proximal and a distal ligature of fine silk have been applied prior to division. *b*, The root is divided between the proximal and distal ligatures (Frazier).

shock from the escape of cerebrospinal fluid (Guleki[18]). It has the disadvantages of not being applicable to the lumbosacral region, because of the difficulty of separating the dura from the vertebrae and the necessity of making a wide exposure in order to attack the roots. The dangers of infection and those relative to leakage of fluid are easily taken care of. The intradural method affords an ample field for identification of the roots with a minimum of trauma to the tissues.

Intradural rhizotomy is the method of choice at this writing. After the cord is exposed, as in laminectomy (p. 1472), *the roots are identified* by their regional arrangement, not always an easy matter. Frazier,[14] as a substitute for cutting the root, uses "root ligation," solely as a means of securing hemostasis. He believes that the presence of even small quantities of blood in the dural sac plays a part in the formation of adhesions, a belief which the writer shares. Figure 733 conveys an excellent idea of the manipulations after the cord is exposed. The cord must be retracted a little to one side or the other, but this retraction must be gentle and never enough to traumatize the roots. As a prophylactic against the harmful effects of trauma, Frazier[14] advocates the application of stovain to the cord and roots just above the area of manipulation. The method is supported by Waugh.[19]

The *after effects in rhizotomy* consist primarily in shock; surviving this, the patient complains of *pain*, which is referred to the distribution of the divided roots and which persists until degeneration is complete (three to five weeks). *Anesthesia* is more or less absolute or extensive according to the number of roots sectioned. *Trophic disturbances* do not occur, if every third or fourth root be left intact. In so-called Little's disease, Clark and Taylor[20] have observed transitory trophic disturbances. In a few instances, the operation has been followed by temporary motor paralysis.

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CHAPTER VII

LAMINECTOMY — CHORDOTOMY

The removal of the spinous processes and laminae, so-called "laminectomy," is performed for purposes of *exploration,* for the treatment of *injuries to the spine and cord* or of *inflammatory lesions* of the spinal canal and its contents, such as lues, actinomycosis and tuberculosis, for the removal of *tumors* and *cysts,* and for operations upon the *spinal roots.* The *technic* of the procedure is easy. The *position of the patient* is important. Figure 734 shows how the patient is postured for exploration of the cervicothoracic region and it is not greatly modified when the lower segments of the spine are attacked. *Endotracheal insufflation ether narcosis* is desirable; local anesthesia may be resorted to (Heidenhain[1]). The instruments employed for the purpose are those shown in connection with operations on bones and joints (Fig. 332), in addition to which, heavy bone cutting forceps should be available. The special equipment consists in a strong self retaining retractor (Fig. 735), Horsley's wax (p. 42), mosquito forceps, and small blunt hooks for picking up the spinal roots (Frazier[2]).

The *incision* is similar to that employed in connection with osteo plasty of the spine (p. 1443); it should be ample and should allow of free exposure of the parts to be attacked. The separation of the muscles from either side of the spine and from the laminae is readily accomplished with the chisel used for splitting the spinous processes for bone grafting. After the *bleeding* is controlled by hot saline gauze tamponade, the self retaining retractor is introduced, the interspinous ligaments are divided, the spinous processes are removed with bone forceps (Fig. 735), and the corresponding laminae with rongeur forceps.

The subsequent steps of the operation depend upon whether an extradural or intradural problem is to be dealt with. Before the dura is opened the thin layer of fat, together with the plexus of veins, is displaced to either side. The dura is next lifted by means of fine silk

sutures, opened with a small slender knife, and held open with the silk traction sutures shown in figure 736. From now on the steps of the operation depend upon the nature of the lesion encountered. The dura is closed with 00 silk introduced with a small curved iris needle, the muscle and fascia are approximated with catgut, and the skin closed with silk or silkworm gut.

FIG. 734.— POSITION OF THE PATIENT ON THE OPERATING TABLE FOR EXPLORATION IN CERVICOTHORACIC REGION.

For adequate exposure of this region a suitable head rest is necessary, in order that the head and neck may be properly flexed. The apparatus attached to this table is capable of adjustment in any direction or angle (Frazier).

The *after treatment* following laminectomy must take into consideration various conditions that do not pertain to operations elsewhere. The patient is at once placed upon a water bed. Fixation is necessary when the cervical region has been operated on; this is accomplished with sand bags. In operations upon the lower sectors of the spine, the possibility of contamination by the excreta must be taken

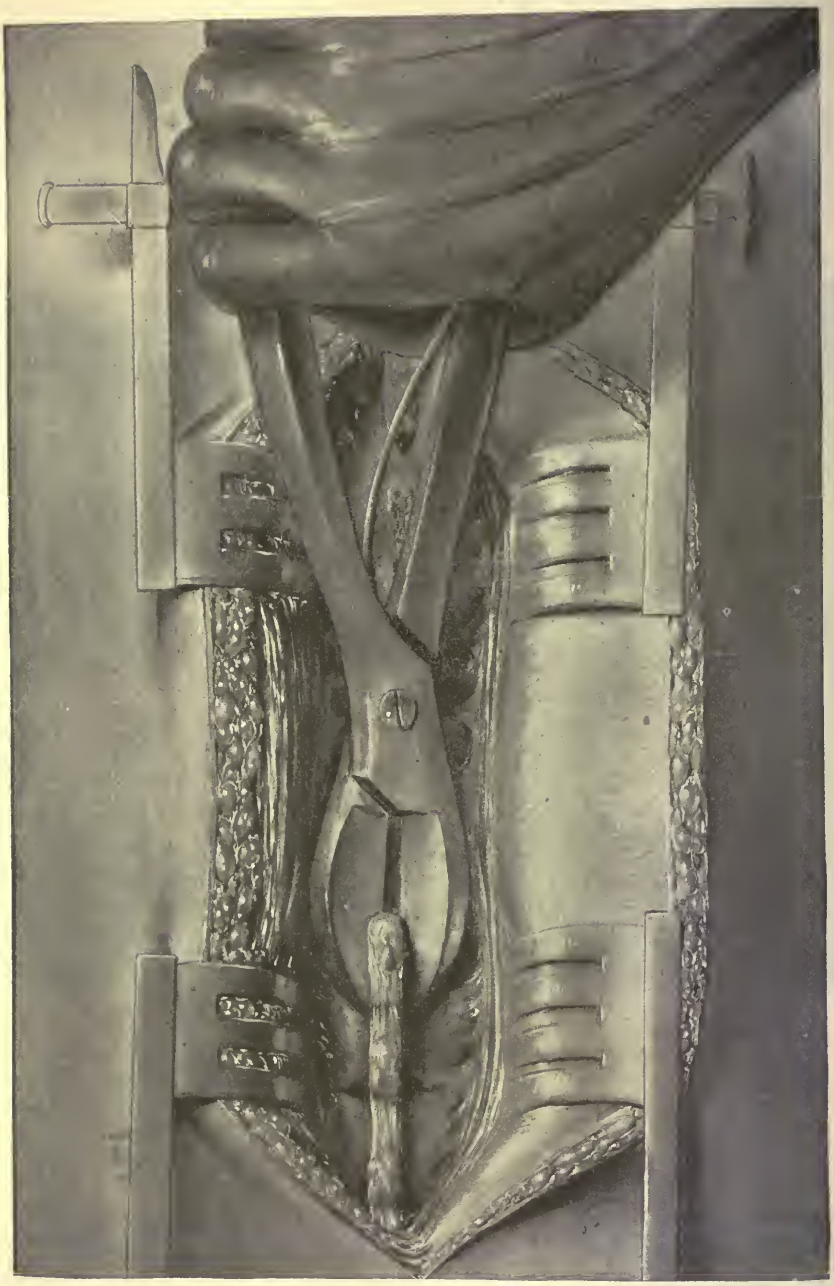

FIG. 735.—TECHNIC OF LAMINECTOMY. Removal of the spinous processes (Frazier).

into account. If there has been much leakage of cerebrospinal fluid, intense headache and vomiting are very likely to occur. In these

FIG. 736.— TECHNIC OF LAMINECTOMY.

The appearance of the operative field after the dural flaps have been reflected with traction sutures (Frazier).

cases the foot of the bed is raised. The liberal use of morphin is permissible, as pain, in some instances, is very severe. The bladder and rectum must receive especial care. Frazier[2] employs suprapubic drain-

age of the bladder in cases in which this organ has been paralyzed for a long time. Frazier[2] furnishes a remarkably clear presentation of the technic of laminectomy. In addition to this, the works of Elsberg,[3] Krause,[4] Schmeiden,[5] and Thorburn[6] may be consulted to advantage.

Decompressive Laminectomy.— As an operation for the relief of pressure upon the spinal cord, laminectomy is often followed by relief of symptoms.

In *tuberculous spondylitis* it is effective in relieving the pressure symptoms due to *angulation,* pressure upon the cord by a *tuberculous exudate,* and pressure from a *displaced sequestrum.* Of the cases operated upon, Lloyd[7] collected 154, 15 of which he did himself. To be of service the operation must be performed before degenerative changes take place in the cord.

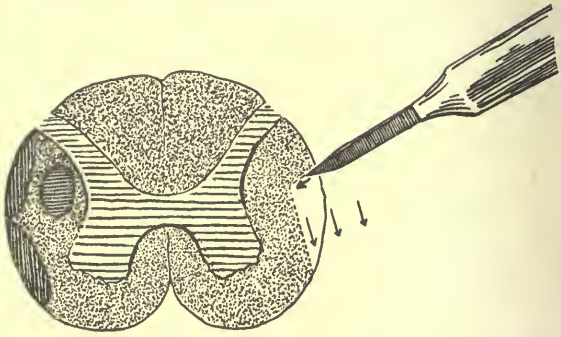

FIG. 737.— DIAGRAMMATIC REPRESENTATION OF SECTION OF AN ANTEROLATERAL COLUMN OF THE CORD, SHOWING THE POINT AT WHICH THE CATARACT KNIFE IS INTRODUCED AND THE DIRECTION IN WHICH THE INCISION IS MADE.

The shaded areas on the opposite side of the cord represent the anterolateral column, the direct cerebellar tract and the crossed pyramidal tract (Frazier).

In *fractures and dislocations* the removal of pressure has a beneficial effect, especially when due to the formation of reparative exudate.

In *inoperable tumors* the measure relieves pain (see Spinal Tumors, p. 1464).

For the relief of *meningomyelitis,* the first decompressive laminectomy was performed by Krause in 1911; four additional cases are reported by Taylor and Stephenson.[8]

Chordotomy.—Chordotomy is a term applied to an incision into the cord for the relief of various conditions. The procedure was suggested by Schüller[9] as a substitute for division of the spinal roots in cases of *gastric crises* and spasticity; by Spiller[10] in cases of unbearable *pain in inoperable tumors* of the cord; and by Krause[4] and Taylor[8] in cases of *meningomyelitis.*

The *operation* (after two or more spinous processes and laminae are

removed) consists in exposure of the cord through a dural slit; then, with a cataract knife, the anterolateral columns of the cord are cut in the manner shown in figure 737. The operation is of recent origin; its execution presupposes thorough knowledge of the anatomy and physiology of the cord and a clearly demonstrated clinical justification for the procedure. For additional information see Allen,[11] Cadwalader,[12] Rothman,[13] Exner,[14] and Beer.[15]

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PART X

SURGERY OF THE HEAD

CHAPTER I

SURGERY OF THE SCALP AND CRANIUM

THE SCALP

Wounds of the Scalp.— Wounds of the scalp present a few regional peculiarities. Force, striking perpendicularly, produces *simple wounds,* striking *obliquely,* wounds with the *formation of flap,* or, if an entire portion of the scalp is torn off, wounds with *loss of substance* (*partial or complete avulsion of the scalp*). Simple wounds of the skin do not gape, those involving the aponeurosis do. Contusions of the scalp are likely to be attended with the formation of a *hematoma.* When these effusions are located beneath or in the skin, they are more or less circumscribed; if beneath the aponeurosis, they may spread over the entire calvarium.

Hematomata beneath the aponeurosis vary with their size; the larger they are, the softer their consistence, the edges being hard, so that *the impression of a depressed fracture of the skull* is likely to be conveyed to the palpating finger.

The *special treatment of wounds of the scalp* relates to *hematoma* and *loss of substance.* As the former is usually absorbed it may ordinarily be left undisturbed. However, if the effusion is very extensive, aspiration may be employed. Incision is resorted to only when pressure necrosis threatens or infection develops. In *avulsion of the scalp,* plastic methods of repair are often necessary. The great vascularity of the scalp and the presence of numerous hair follicles make the scalp pecularily resistant both to trauma and to infection, so that repair by suture of a flap left attached by only a slender pedicle is often followed by healing. A useful method of repair in connection with avulsion of the scalp is shown in figure 738.

Traumatic emphysema of the scalp occurs in connection with the establishment of a subcutaneous opening into the air sinuses of the cranial bones, most frequently after fracture of the frontal bone involving the frontal sinus, and at times the mastoid process. It usually disappears spontaneously (see Traumatic Emphysema).

Pneumatocele of the head is due to the same causes as traumatic emphysema, but the air is located beneath the periosteum. The collection of air raises the periosteum into a tense, more or less circumscribed, elastic *tumor* which may be reduced when the contents are forced into the sinus it communicates with by palpation, and increase in size during forced expiration. The communication with the frontal or mastoid sinus may be *congenital* or may be established by *caries* or be the result of *trauma*.

The *treatment of pneumatocele cranii* consists in exposure of the opening and removal of carious bone — when present — and closure of the defect in the bone by *osteoplastic methods* (p. 1566).

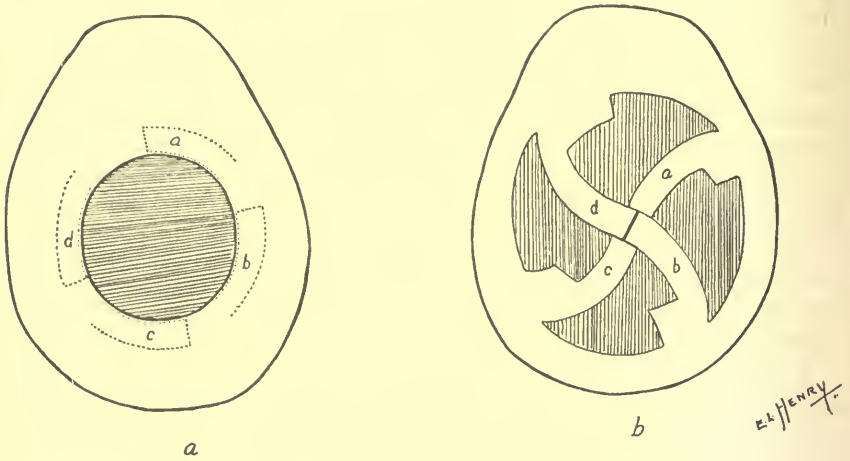


FIG. 738.— PLASTIC REPAIR OF SCALP.

Tumors of the Scalp.— Lipomata (see p. 1134) of the hairy scalp are rare; they are likely to be very vascular. The growth is differentiated from dermoid cysts by the invariable location of the latter. The *treatment* consists in excision, usually not a difficult matter, although control of bleeding may be troublesome.

Fibromata (p. 1119) *of the scalp* usually appear in papillomatous form. Four types may be distinguished, (1) *soft multiple fibromata* —fibromata mollusca — which originate in the sheaths of the cutaneous nerves; (2) the pigmented "*neuronevi*," in which regular, minute fibromata are attached to the terminal twigs of the cutaneous nerves; (3) the "*racemose neuromata*," in which coils of the thickened nerve fibers — cylindrical or spindle shaped — are twisted and intertwined

like grapevines; and (4) elephantiasis of neuropathic origin, or *elephantiasis neuromatosa congenita.* The four may exist in combination with one another.

Treatment by excision is complicated with the danger of bleeding, which is likely to be menacing. It may be necessary to remove the growth in sections. When the process is extensive, plastic repair of the denuded area is necessary.

Angioma frequently develops in the scalp. It occurs in three forms, *angioma simplex, angioma cavernosum,* and *angioma arteriale racemosum* (see p. 1158).

Angioma simplex (telangiectasis or nevus vasculosus) appears most often upon the face and is usually situated on the forehead in the region of the glabella and the inner portion of the eyebrows, though it also appears at the fontanelles, sutures, parietal eminences and frontal tuberosities. Telangiectasis may occur alone or in combination with other forms of angioma and with lymphangioma.

The *treatment* with caustics or by excision is described above (p. 1163).

Cavernous angioma, as already stated (p. 1161), is composed of a spongy mass of blood filled sinuses. They not infrequently communicate with the longitudinal and transverse sinus.

Excision is a desirable method of treatment. However, when it is evident that the tumor communicates with one of the large cranial sinuses, it is best to employ *multiple puncture with the thermocautery.*

Racemose arterial angioma (cirsoid aneurism), described on page 1164 (Fig. 566), frequently occurs on the scalp, and usually develops in the course of a large vessel communicating with the carotid artery. The *diagnosis* is not difficult and is discussed on page 1166. This form of angioma must be differentiated from pulsating sarcoma, from arterial aneurism, and from arteriovenous aneurism.

The *treatment* is discussed on page 1166. Of the various methods that of *ligature of the afferent arteries and total* extirpation is the least unsatisfactory. In fact, if this is done early in the process, the result is likely to be gratifying.

Aneurisms (p. 976) of the scalp are infrequent. *Simple aneurism* of arteries supplying the scalp is almost always caused by some injury of the vessel. The *superficial temporal,* because of its extensive distribution and its superficial course, is the one most exposed to violence and is most often the seat of aneurism. The aneurism may be *sacculated* or *spindle shaped.* The *diagnosis* presents no difficulties.

The *treatment* of aneurism in this situation consists in extirpation of the sac.

Arteriovenous aneurism (p. 981) also occurs most frequently in the region of the *superficial temporal artery* and is the result of injury to this vessel, usually in connection with a punctured wound. The most conspicuous *symptom* is dilatation of the superficial veins.

The *diagnosis* is taken up on page 986.

The *treatment* consists in double ligature and excision of both artery and vein (for extended discussion see p. 990).

Sinus pericranii is a peculiar cyst-like growth filled with blood, situated beneath the cranial periosteum and is connected with the longitudinal sinus by a wide emissary vessel. It has some of the characteristics of subcutaneous cavernous angioma. The lesion is *probably traumatic in origin* and is situated in the middle of the skull, on the forehead, or on the occiput. It is covered with normal or slightly atrophic skin and is rarely larger than a walnut. It is *soft and elastic,* and may be forced into the hollow of the skull, where a cleft may be felt in the bone or a bony ring about the base of the tumor. The growth increases in size and becomes tense when the head is lowered, or when the patient coughs or sneezes.

The *treatment* by continuous pressure is not usually successful. In one case Krause[1] exposed the growth by lifting the skin from it in the form of a flap, and drilling the bone contiguous to the emissary. Through this opening he tied the emissary, excised the now bloodless tumor and closed both openings in the skull by means of an osteoplastic flap fashioned from the contiguous skull. The method is described in detail and the steps of the operation are shown in a series of illustrations (see bibliography No. 1).

Sarcoma (p. 1172) of the scalp occurs either as sarcoma of the skin or of the fascia. The first originates in the intact connective tissue of the skin or subcutaneous tissue, or develops secondarily to some disturbance in the normal structure of the tissue, as from keloid, warts or pigmented moles. Its *clinical course* does not differ from that of sarcoma in other parts of the body. There are two types, *soft nodular tumors, and warty growths.* The former may be elevated above the level of the skin and have overhanging mushroom-like elges.

The *melanotic sarcomata* (p. 1200), developing secondarily in small pigmented moles or warts, have a particularly malignant character. They occur as diffuse nodular growths or in the form of numerous hard knots and have a tendency to form early metastases.

In *sarcomatous warts,* on the other hand, the outlook is less unfavorable. When these are excised they show little tendency to recur.

Because of the great vascularity of the scalp, sarcomata are very likely to be of the pulsating variety.

Sebaceous cysts (atheromata) are the most frequent form of tumors of the scalp. Their structure is not different from that of cysts of the same character located elsewhere on the body. They are very likely to be multiple, and at times reach great size. Small cysts are hard, large ones are always soft. The latter usually fluctuate. The superimposed skin is thin and pale. Very often the summit of the tumor shows pitted hair follicles destitute of hair, or it may be the seat of a comedo. Their structure and contents are described on page 1316. Occasionally a sebaceous cyst of the scalp becomes malignant.

The *treatment* consists in excision *in toto.* If a portion of the sac is left, the growth returns.

Dermoid cysts (p. 1237) occur on the head, as they do elsewhere on the body, before the second or third year of life. The skin over them is unaltered. They are found beneath the skin and below the aponeurosis of the occipitofrontalis muscle. In some instances such a cyst is so intimately associated with the periosteum that a sector of this membrane must be removed with it.

Dermoid cysts are distinguished from other cystic and solid tumors by their *deep situation* and their *constant location.* They are found in the neighborhood of the anterior fontanel at the outer edge of the *supra-orbital margin* and the contiguous *temporal region;* also at the *inner angle of the eye,* over the *glabella* and the *root of the nose.* These situations correspond to the points of union of the embryonic clefts.

The *treatment* consists in *extirpation* of the growth. This is especially important in children, in whom the tumor interferes with the normal development of the contiguous bones.

Carcinoma (p. 1253) of the scalp originates from the skin. It does not present any regional peculiarities, except perhaps that its growth is likely to be very slow. Its development in connection with sebaceous cysts should be borne in mind. In the treatment, the accessibility of the growth and its slow progress may tempt the surgeon to employ caustics and "light therapy." *Early and complete removal is the correct treatment* and when thoroughly executed, there is little likelihood of a return of the growth.

Inflammations of the Scalp.— Erysipelas (p. 264) occurs in the scalp

more often than in any other portion of the body. It often arises in connection with *suppurative otitis media* and during its onset is *frequently mistaken for mastoïditis.*

The *temperature* in erysipelas of the scalp is *very high* and is attended with delirium, semistupor, and muscular twitching formerly regarded as indicating meningitis. *Meningitis* in connection with erysipelas of the scalp occurs when streptococcus infection extends to the cranial cavity through the site of a fracture or by means of an osteomyelitis traversing the bone, and also by extension of thrombosis of the veins of the scalp through the emissaries to the longitudinal sinus.

Abscesses of the scalp are a frequent *sequel* of erysipelas. Among the most serious are those in the adipose tissue of the orbital cavity, whence they may extend to the brain. In many instances temporary alopecia occurs.

As a rule, the *prognosis* is good; it depends largely upon the complications.

The *treatment* is purely symptomatic.

Phlegmons of the scalp are either *circumscribed or diffuse.* The *circumscribed* form appears as a *furuncle* or *carbuncle* (staphylococcus). The *diffuse phlegmons* (cellulitis purulenta), on the other hand, are usually caused by the streptococcus family and are likely to involve extensive areas of the scalp. As they are situated both *above and below the aponeurosis,* they frequently cause necrosis of the latter. In not a few instances, the *external table of the skull is destroyed.* The process involves great danger of extension to the brain, especially in infected punctured wounds.

The *treatment* consists in early, free, *deep incisions* through the aponeurosis and periosteum to the bone. The incisions should be planned to provide for *free drainage* at the supra-orbital ridge, the mastoid and the occiput. Light tamponade with iodoform gauze is permissible for a few days only.

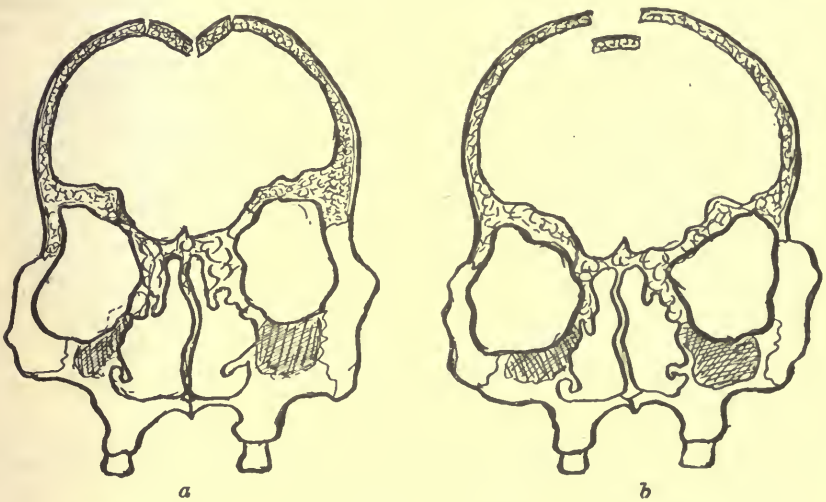
THE CRANIUM

Fractures of the Skull.— Fractures of the skull, as such, have no particular significance; their importance pertains to their relationship to the brain. The outcome of injuries of this class depends very little upon their form and extent at the time, but relates to the trauma inflicted upon the brain and its vessels. There are portions of the brain which bear trauma without much disturbance of function, while at

the base comparatively slight injury becomes a serious matter. Fractures of the skull may be divided into those of the *vault and those of the base.*

The *various forms of fracture of the skull* do not differ from those described in the chapter on fractures (p. 639). However, the conformation of a fracture in this situation possesses *peculiar clinical importance.*

Fissured Fractures.— Fissured fractures appear in the form of cracks or splits. They occur as the result of a bursting or bending force (see mechanism of fracture of the skull, p. 1489). As a rule, the edges do not gape. They may be *single or multiple, simple or forked,*



a *b*

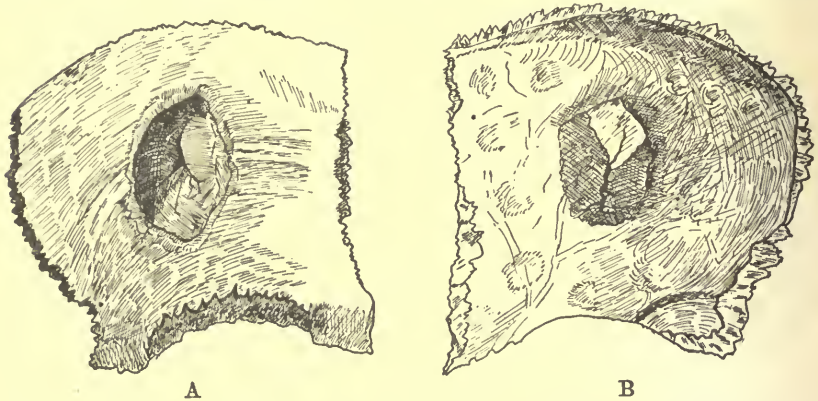
Fig. 739.— *a,* Central Depression; *b,* Peripheral Depression.

or divided into many branches, and may be limited to one bone or may involve several. They may be short or long, straight or curved. They often cross the lines of the sutures, though they are likely to change direction when this occurs. Finally, fissured fractures often occur in connection with penetrating gunshot fractures, with splintered and comminuted fractures, and with those attended with loss of substance.

Comminuted Fractures.— Fragmented, comminuted and splintered fractures are common. In the *fragmented fracture* there is only one line of fracture. It encircles an area of the skull in a curve or in an angular line separating its connection with the rest of the skull. It is the outcome of a bending force. In *splintered or comminuted*

fractures a greater or less proportion of the skull has been broken into a number of pieces. The splinters may or may not be separated from the soft parts. At times the arrangement of the fragments justifies the use of the term *stellate fracture.*

The degree of displacement in fracture of the skull has an important bearing on the outcome, especially when the fragments encroach upon the cranial cavity. Accordingly, FRACTURES are divided into those WITH, AND THOSE WITHOUT DEPRESSION. The entire fragment circumscribed by the line of fracture may be depressed (*peripheral depression*) (Fig. 739). In this case the external table of the depressed center is usually fissured, while the internal table, still more depressed, is always more extensively separated. Figure 740A shows



A B

FIG. 740.— *A*, FRACTURE OF THE OUTER TABLE OF THE SKULL; *B*, THE SAME FRACTURE FROM WITHIN.

the outer and B shows the inner aspect of a fracture of this part. At times the depressed disk of bone is either broken into many fragments or is only slightly cracked; at others, the fragments are pushed over one another, or under the adjacent edge of a fracture between bone or dura, or are displaced laterally. They may penetrate the dura and become embedded in the brain. It is these possibilities that make splintered fractures a source of great danger to the patient.

PERFORATED FRACTURES— *Perforated fractures and fractures with loss of substance* are the result of gunshot wounds of the skull or are caused by penetrating hooks, or falling spikes, or both. The mechanism of gunshot wounds is discussed farther on. Extensive loss of substance is caused by shell fragments. In comminuted fractures

in which the skull is broken into many pieces, some of the fragments are entirely separated, tearing extensive openings in the vault of the cranium. The various forms of fracture involve either both tables to the same extent, the inner table more than the outer (as is usually the case), or only one table. Under the latter circumstances, the inner or outer table may be involved alone. The outer table may show little, if any, injury, yet the inner (lamina vitrea) may be splintered—the so-called *isolated fracture of the inner table.* Fracture of the outer table alone occurs, but is rare. This is important, as even a SLIGHT FISSURED FRACTURE OF THE OUTER TABLE IS ALMOST CERTAIN TO MEAN SOME SPLINTERING OF THE INNER.

In the *mechanical processes involved in fracture of the skull* it must be remembered that two things determine the elasticity and rigidity of the skull, the character of the material (the bony tissues entering into its construction) and its shape. The *elasticity of the bony tissues* is that quality by which these tissues resume their original form when distorted by stress or strain. This quality has been placed on a mathematical basis by Rauber.[2] The distortion produced by violence caused by a broad smooth surface is not limited to a circumscribed portion, but produces a change in form of the entire skull. If, in a fall, the skull strikes a broad surface, or is struck by a blow from a broad surface, the vertical diameter is shortened, while at the same time the occipitofrontal and biparietal diameters are increased. The particles in the direction of the increased diameters are drawn apart, and those in the diminished diameters are pressed together, until at last the skull breaks or, more properly speaking, *bursts.*

As the skull is elastic, the lines of fracture must gape when the skull is burst or fractured, but as soon as the violence ceases to act they return to their original position and become closed. This explains how orbital fat is caught in fissures in the roof of the orbit; also, dura mater and hairs. Considering the elasticity of the skull, we may divide fractures into two forms, *bending fractures and bursting fractures.* These include all fractures of the vault and base, even the fissured, comminuted and perforated. The mechanism of bending fractures is more clearly defined in those produced by objects and instruments of relatively small surface; that of bursting fractures, in those produced by violence caused by extensive surfaces. Whenever the skull is compressed in any given diameter, there is a shortening in the direction of pressure, while the diameters of the circles perpendicular to this line of compression are increased. The FORM OF THE

SKULL IS CHANGED IN THESE DIRECTIONS BY EVERY VIOLENCE, WHETHER ACTING ON A LIMITED OR CIRCUMSCRIBED AREA OR MORE EXTENSIVELY. According to the extent of the surface possessed by a striking body, an area of skull is flattened, then depressed and bent in. If the action be momentary, as in a blow, and its force exhausted by the elasticity of the depressed portion of the skull before its limit of elasticity has been reached, the cranium rebounds to its former position and no permanent change results. The bending, however, results in fracture when the molecules of the skull have reached a degree of separation in which the force of cohesion no longer suffices. This takes place first in the inner table, the solitary fracture of which is thus explained. The mechanism of this fracture may be illustrated by observing the way a stick breaks when bent over the knee, i. e., the fracture begins on the side opposite the causative force.

At the site of a dent in the skull there are always found cracks and fissures and, in the majority of cases, comminution and splintering (except in the skulls of infants).

While "bending fractures" are the result of violence immediately at the site of impact, BURSTING FRACTURES OCCUR IN RESPONSE TO A CHANGE IN THE FORM OF THE ENTIRE SKULL. By the combination of bending and bursting fractures, those lines of fractures are explained that surround extensive comminuted fractures and radiate in many directions. The irregularity in thickness of the various sectors of the skull renders the establishment of a definite mathematical rule in this connection impossible.

Bursting fractures in combination with bending fractures are best studied at the base. The greatest degree of tension sustained by the flattened meridian circles is at their midpoint where they cross the equator of the sphere. At this place bursting should begin in each instance. As, however, the base of the skull possesses greater rigidity than its convexity, the bursting almost always occurs in the basal portion of the meridian. Compression from side to side produces the greatest degree of deformity and readily causes the skull to burst. This is observed when a wagon wheel passes over the head, the fracture being usually a bursting, transverse one through the base, *complicated by a bending fracture of the vault on the side receiving the impact.* In a certain sense, compression of both sides of the skull also takes place in falls in which the head is struck. In this case the skull strikes the ground, but the spinal column following after, is driven against the condyloid processes of the occipital bone with the weight of

the body and the velocity of the fall. The atlas striking against a circumscribed area of skull brings about the lateral circles of a bent-in fracture (*ring fracture*).

Isolated short fractures of the base are incomplete bursting fractures. They occur in the orbital plates, usually on both sides at the same time.

Besides bending and bursting, *expansion* must be mentioned as a factor *in the production of fracture of the skull.* This is taken up more extensively in connection with gunshot fractures (p. 914).

The *diagnosis of fracture of the skull* before the advent of the Röntgenogram was a much more difficult matter than it is at this writing. With the technical refinements of modern Röntgenology, few fractures of the skull, including those of the base, escape recognition.

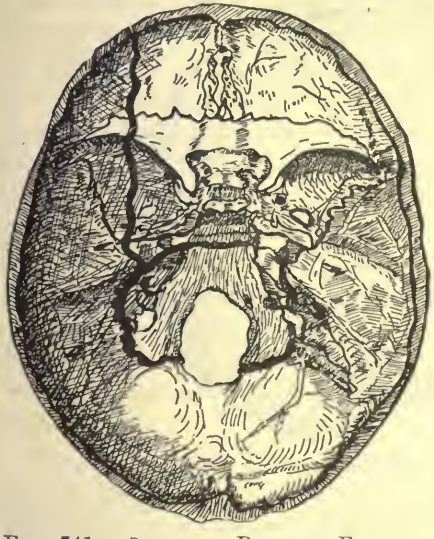


FIG. 741.—CIRCULAR BENDING FRACTURE OF THE SKULL, WITH LINEAR BURSTING FRACTURE EXTENDING INTO THE LEFT ANTERIOR FOSSA.

Though important as regards *prognosis,* cerebral manifestations usually accompanying fractures are of little aid in making the diagnosis of fracture. In *simple fractures* palpation of the surface of the skull is of aid in discovering a fissure, the edge of a fracture, or *an area of depression,* but this is only possible when the last is of considerable depth. Extensive comminution of bone is more easily determined in this way. In this connection must be excluded (1) depressions produced in the course of parturition and by previous injuries; (2) senile atrophy of the skull; (3) the hollows due to lues; (4) the natural prominences and irregularities, the result of peculiar cranial conformation. Difficulties in examination arise in connection with contusions of the soft parts. Here flat hematomata with a hard firm base often lead to error. The diagnosis of subcutaneous fractures (except by X-ray) can be made with certainty only in those cases in which there is displacement toward the cranial cavity, or in which fragments have broken loose. Fortunately the welfare of the patient is rarely depend-

ent upon the positive diagnosis of subcutaneous fracture. At the present time, the exposure of an area of the skull by incision for the purpose of diagnosticating fracture is not practiced. In cases of subcutaneous fractures of the skull, operative interference is necessary only for relief of accompanying lesions of the cranial contents, not because of the fracture itself.

When there is a wound of the soft parts and its communication with a fracture is suspected (compound fracture), this may be determined

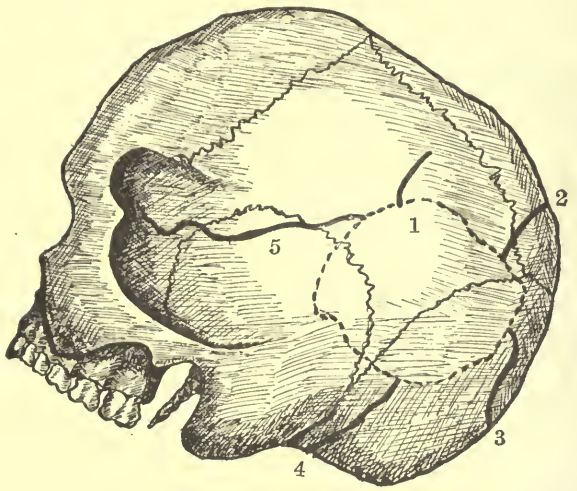


FIG. 742.—BENDING AND BURSTING FRACTURE.

Bending fracture outlined by dotted line; bursting fracture by continuous line. Fracture caused by bilateral compression; point of impact the occipital region. Poles connected by bursting fracture (4), which begins on the vault at the bending fracture and passes through the posterior fossa, then through the clivus, sella turcica, and roof of the orbit. The bursting fracture (2) passes over the left parietal to the external angular process of frontal; the fissure (3) has a short course, while (5) passes over the right parietal to the great wing of the sphenoid and ends in the sphenotemporal suture.

by the sense of touch and sight. For the purpose, the exploration is made under aseptic precautions, with the patient narcotized. If these conditions are not possible, it is best not to meddle with the injury, but to cleanse and treat it as described in connection with wounds in general (p. 3).

The question of *injury to the dura* is of great prognostic importance. This is determined during the examination as stated above; the extrusion of brain substance of course makes this certain.

The *symptoms of fractures of the base* are indirect. The points in their diagnosis are as follows:

1. The spreading of hemorrhages from the site of fracture to certain points under the skin, where they appear as ecchymoses.

2. The flow of brain tissue, blood, and serous fluid from those cavities contiguous to the base.

3. The disturbances in function of the cranial nerves emerging at the inferior surface.

Extravasations of blood appear where the skin and mucous membranes are attached to the base of the skull by loose connective tissue. These sites correspond to the *eyelids* and the connective tissue of the *eye,* the mucosa of the *pharynx,* the *region of the mastoid process,* and the *sides of the neck.* Their appearance, however, is positive evidence of fracture only when the site of the injury is at some distance from them and the extravasation does not immediately follow the trauma. The so-called "black eye" is especially misleading, and is of diagnostic value only under the conditions stated. *Exophthalmos* occurring immediately or soon after violence, followed by ecchymosis of the conjunctiva and eyelids, is an indubitable sign of orbital hemorrhage and of fracture of the base. *Ecchymosis* into the mucosa *of the pharynx* is rarely observed. The dense connective tissue in this situation does not favor its extension. *Ecchymosis* in the region *of the mastoid process* indicates a fracture in the squamous portion of the temporal bone.

The *flow of brain tissue,* from clefts of bone produced by fracture, through the divided soft parts is a positive sign of fracture of the base and, at the same time, of injury to the membranes of the brain. This has been observed at the external auditory canal and the nose; the latter is uncommon.

The *hemorrhages from cavities and canals* that adjoin the base take place from the *ear,* the *nose,* and the *pharynx.* The flow of blood from the ear possesses the greater diagnostic value. It escapes through the bony canals and fissures in the petrous portion of the temporal bone.

Hemorrhage from the ear may originate from (1) bursting and laceration of the ear drum; (2) fracture of the anterior wall of the external auditory meatus; (3) violent separation of the cartilaginous and bony portions of the auditory canal; (4) a fracture involving the mastoid cells; (5) fissures and fractures of the base of the skull. Again, not every fracture of the petrous portion is attended with hemorrhage from the ear; it occurs only when the drum is ruptured.

In these cases the blood gains access to the pharynx by way of the eustachian tube and is swallowed or rejected or flows out through the nose. In the last instance, the blood may come from a fracture of the horizontal plate of the ethmoid.

The flow of serous fluid from the ears in fracture of the base is a symptom of fracture of the petrous bone. The fluid comes from the subarachnoid spaces, proving that the tympanum is ruptured. The flow of fluid may take place immediately after the injury. In most cases, however, it does not appear until twenty-four hours later. The quantity of blood is always large, 150 to 200 gms.; even more is not uncommon. When, in cases of fracture of the petrous bone, *the tympanum remains intact,* the fluid may flow through the eustachian tube into the nares. It may flow from the ear and the nose at the same time.

Paralyses of single cranial nerves are produced by fractures that traverse the canal or foramen through which the nerve emerges from the skull. The nerve is either *lacerated, divided, compressed,* or *contused.* The result of the injury is immediate paralysis of the part supplied by it. Besides this, a second form of paralysis develops as a result of an inflammation extending along the nerve trunk; this is usually ascending. The facial nerve is most frequently paralyzed. A number of cranial nerves may be paralyzed at the same time (v. Bruns[3]).

According to Stewart,[4] *Röntgenographic examinations* show that "in over fifty per cent of the cases, both the vault and the base are found fractured. In three hundred cases of head injury, twenty per cent showed fracture of the skull. While objective signs, such as bleeding from the ears, etc., may give a clue to the location of the fracture, these may be misleading and in every case a systematic radiographic technic must be followed." Stewart's[4] presentation goes into great detail in this connection, suggesting a method of procedure in which the entire vault and base of the skull is visible on the negatives. The proper interpretation of the radiogram presupposes a knowledge of the normal sutures and fissures and foramina of the skull. Stewart emphasizes the value of the stereoröntgenogram. This is especially true in linear fractures extending into the roof of the orbit.

In the *course of fractures of the skull,* the bony lesion is not of great importance; the SIGNIFICANCE OF THE INJURY LIES IN THE COMPLICATING INVOLVEMENT OF THE CONTENTS OF THE CRANIUM and in INFECTION.

The dangers of concussion, compression, contusion and laceration of the brain are common to all fractures of the skull; the dangers of infection of the connective tissue, the bone, the meninges, and the development of brain abscess and sinus thrombosis are factors only when injuries of the soft parts communicate with the seat of fracture.

The *involvement of the cranial contents determines the therapeutic problem* (v. Bergmann[5]). This is taken up farther on. The unfavorable factor in fractures of the base relates not to the bone

FIG. 743.—RÖNTGENOGRAM OF FRACTURE OF THE VAULT OF THE SKULL (Harlem Hospital case).

lesion, but to the brain injury. The fracture, of itself, is not susceptible to surgical measures of relief.

The so-called provisional callus of fractures in general is not found in fractures of the skull; this is ascribed to the absence of irritation from muscular movements which determine its development in the former, and to the destructive force of the trauma in the latter. Fractures of the skull are directly *repaired by osseous closure of the fissures and clefts.* This takes place even in the most extensive frac-

tures and includes *loose fragments*. Fractures with loss of substance are followed by residual defects.

The *treatment of fractures of the skull* in cases of subcutaneous lesions is different from that of those complicated by wounds of the soft parts.

Subcutaneous fractures, unless attended with symptoms of involvement of the cranial contents, or with marked depression shown in the Röntgenogram, *are best treated expectantly.*

In COMPOUND FRACTURES OF THE SKULL THE TREATMENT, AS FAR AS THE BONE LESION IS CONCERNED, IS PRECISELY THAT OF A SIMILAR INJURY INVOLVING THE TIBIA. Detached splinters may be removed or placed into the niveau of the skull; moderate depression may be disregarded. The *question of trephining for depressed fracture per se* may be answered as follows: Slight depression cannot possibly be responsible for cerebral compression; the latter may be due to intracranial bleeding coincident with a fracture, but this occurs when there is no bony depression. It often happens that a splinter of bone penetrates the brain and is responsible for focal symptoms, but this is not in any sense a cerebral compression caused by a depressed fracture, and indication for operation is presented by the focal symptoms (restricted spasm and paralysis). The notion that *epilepsy* is caused by the pressure of a restricted area of bony depression and that this contributes an indication for operation is also not tenable. *Epilepsy follows head injuries without fracture* and is due to a lesion of the brain or its coverings and subsequent scar formation. This is explainable on the basis that in these injuries the trauma has not exhausted the elasticity of the bone, that the latter reassumes its normal outline and the contused brain area underneath becomes sclerosed and is responsible for so-called jacksonian epilepsy. The removal of the depressed area of bone does not prevent the subsequent development of the latter (Küttner[6]).

In the *treatment of compound fractures,* the wound is cleansed, splinters of bone are removed and large fragments are placed in their normal relationship. This is accomplished by free exposure of the injury and is followed by the usual treatment of wounds. The primary cleansing of the wound and its repair are aimed at *prevention of infection,* and are not destined to provide an unusual method of the treatment of fractures. It may well be added that the development of infection in this situation is attended with such grave menace that no precaution in this connection may be omitted. In cases of exten-

sive loss of substance, the question of immediate osteoplastic closure of the defect must be given serious consideration. This is of course permissible only when there is reason to believe that infection has not occurred. However, in most cases, the closure must be postponed until danger in this connection no longer exists. The special dangers of infection pertain to *leptomeningitis purulenta* and to *suppurative infection of the bone.* The latter is treated in the same way as is osteomyelitis elsewhere in the body.

The *treatment of fracture of the base of the skull* aims at the prevention of infection. This consists more in withstanding the temptation to meddle than in active measures of relief. As the infection enters through the cavities of the face and ears, these must be kept clean. The latter may be lightly tamponed with iodoform gauze, the tampon being changed as soon as soiled. Tamponade of the nasal passages is inadvisable. In cases in which the effusion of blood is attended with the clinical picture of cerebral compression, the question of affording an avenue of egress for the fluid must be taken into account. For the purpose, Cushing[7] approaches the middle fossa of the skull by exposing the temporal bone immediately above the zygoma, opens the squamous plate with a burr, and enlarges the opening with a rongeur forceps. The procedure has a certain limited field of usefulness. When purulent meningitis has developed, the employment of the *decompressive craniotomy* must also be given consideration.

Incised, Punctured and Gunshot Wounds of the Skull.— The surgical importance of *incised* wounds of the skull may be divided into (1) simple cuts limited to the outer table; (2) division of both tables; separation of a flap; (4) complete separation of a segment of the skull in which the bone may remain attached to the soft parts, or both may be cut off at the same time. Wounds of this sort may be classified as *penetrating, or nonpenerating.*

These injuries usually run a favorable *course.* The outcome is dependent upon the concomitant *brain injury* and upon the development of infection.

The *treatment* in all essential regards is *similar to that of compound fracture.*

Punctured wounds of the skull are very likely to involve the cranial contents and frequently are infected. The roof of the orbit is especially liable to injuries of this sort (pointed tools, bayonets, arrows, etc.). In not a few instances a portion of the invading instrument is (3) oblique or horizontal splitting of both tables without complete

left wedged into the cleft in the bone. Other than the danger of infection, the possibility of the retention of a foreign body makes this kind of injury a serious one. Verification by *radiogram* is of great help in the *diagnosis*. The *treatment* is also similar to that of compound fracture. In addition to this the foreign body must be removed (see Technic of Trephining, p. 1564).

Gunshot wounds of the skull have been surrounded with an atmosphere of mystery which would not seem justified. As a matter of fact, as far as the surgical problem is concerned, the clinical factors in this class of injury differ from those encompassed by the study of compound fractures in this situation only with regard to projectiles fired at close range, and this relates to the fact that, in addition to the bursting and bending effect of the projectile, the *hydrodynamic action of the fluid constituents of the brain* is operative with regard to the extent of the lesion. This was worked out by v. Color and Schjerning[8] in 1894, and remains unchanged at this writing. Thus, the action of projectiles fired at close range becomes *expansive* in character and is attended with severe injury of the entire brain in the form of diffuse hemorrhages and areas of contusion, the ventricles being filled with fluid blood, which is present even when projectiles traverse only the bone. It would seem permissible to classify gunshot wounds of the skull (other than those fired at close range) with the bending and bursting compound fractures due to other causes, the extent of the lesion depending upon the velocity of the projectile at the moment of impact. That a projectile may strike the skull directly or at an angle goes without saying, and that the resultant lesion would not differ in character from that produced by other agents, such as an ax, a hammer, or a pick, is equally patent. Indeed, it not infrequently happens that the impact of a projectile is so slight that it (just as any other force) does not exhaust the elasticity of the bone at the point of contact, and that the latter reassumes its normal shape without fracture, as already described in connection with simple fractures (p. 1489).

Gunshot wounds, like punctured wounds, may also be divided into *penetrating and non-penetrating*. To this may be added the fanciful class of *penetrating gutter wounds* of the skull in which the bullet plows a furrow in the bone; yet these injuries may be very properly classed with wounds of the skull with restricted loss of substance.

The *treatment of recent gunshot wounds* of the skull does not differ from that of similar wounds of the extremities and joints. Apart from rapidly increasing cerebral pressure, the element to be most feared in

gunshot wounds is *infection*, and this pertains especially to the cranial contents. Aside from the first aid dressing, discussed elsewhere (p. 57), the question as to whether wounds of this sort should be closed at once, or treated as infected wounds and left open, has occupied the attention of surgeons for many years, and with widely divergent conclusions. Especially in the recent war has there been a tendency to practice the former method, though this is not by any means universal. If the factors adduced in this connection (p. 1489) are based on the proper conception of the "mechanism of infection," then gunshot wounds should never be closed, but should be treated by the open method. In perhaps the most recent work pertaining to this class of cases, Chatelin and De Martel[9] say "The surgeon must be satisfied with facilitating, with judicious drainage, the elimination of all dead material, the presence of which provokes and encourages infection. Men wounded in this way never die of osteomyelitis; that is not the danger. The immediate danger is, almost exclusively, meningitis, and the natural defenses of the wounded man are much more effective against it than is the illusionary help of surgery."

Diseases of the Skull.— Rachitis of the skull (craniotabes) is a part of a general rachitic condition characterized by persistence of the fontanels and the soft consistency of the bones, especially of the occipital, which cause it to become flattened (tête carrée). It may be mistaken for congenital hydrocephalus. For the treatment see page 1503.

Osteomalacia, chronic osteitis deformans, and osteoporosis of the skull are uncommon.

Tumors of the Cranial Bones.— Osteomata of the skull are usually situated on the frontal and parietal bones. They may be located on the external or internal surfaces of the skull or on both, and are often multiple. Traumatic origin has been claimed, but not proved. *Exostoses of the orbit* are of especial interest. They are usually located in the labyrinth of the ethmoid bone. Their development has been studied by Bornhaupt.[10] The *diagnosis* rests on their slow growth and the Röntgenogram. They must be separated from chronic inflammatory lesions of the bone, especially lues. They should be *excised*.

Sarcoma appears primarily in the cranial bones. Its clinical course is similar to that of sarcoma in other portions of the body. Sarcomata in this location grow very rapidly and metastasize early.

Sarcomata of the dura present the clinical picture of brain tumor

until they perforate the cranium. The former is at first attended with brain irritation and later, compression.

The *diagnosis of cranial sarcoma* must take into account that primary carcinoma does not occur in this situation. Recognition of the nature of the growth rests upon the examination of a microscopical section and (by exclusion) on the Röntgenographic findings. The *treatment* consists in complete removal of the growth. The *prognosis* is unfavorable (Wrede[11]).

Lues of the Bones of the Skull.— Luetic lesions of the skull in children are *hereditary* (Darrier and Feulard[12]). In the acquired disease the lesions of the skull occur in the *tertiary stage*. They occur as (1) *gumma of the periosteum*, and (2) *gumma of the diploë*, and are often attended with a purulent mixed infection. The *clinical course* of the disease does not differ from that of bone lues elsewhere in the body.

Aside from the *constitutional treatment*, removal of the diseased bone is made imperative because of the danger of accidental mixed infection and its extension to the cranial contents. As in all gummata, removal of the focus is followed by a marked improvement in the general condition of the patient. Regeneration follows resection of even extensive areas of bone (Hofmeister[13]).

Tuberculosis of the Skull.— Tuberculosis of the bones of the skull is not uncommon. It is usually manifested by the *clinical picture of a cold abscess* and, though it occurs in the adult, it is more frequently found in children. Krause[14] and Wieting and Raif Effendi[15] furnish instructive presentations on the subject. The disease appears in the *circumscribed* and in the *infiltrating* forms. The possibility of the development of the latter form indicates the treatment, which (aside from the constitutional care) consists in excision of the diseased bone. In extensive processes this may have to be supplemented by osteoplastic repair of the residual defect (see Bone Tuberculosis, p. 441).

Acute Osteomyelitis of the Skull.— Recognition of the dependence of acute osteomyelitis of the cranial bones upon the same factors which provoke it in the long bones of the body is due to the work of Lexer.[16] The *clinical course*, etc., of the disease is taken up in detail in Part II, chap. x. Its especial significance in the skull relates to the danger of extension of the infection to the cranial contents. This makes the prompt employment of operative measures of relief imperative.

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CHAPTER II

THE SURGERY OF THE BRAIN, ITS MEMBRANES AND VESSELS

Cephalocele.— Cephalocele are congenital tumors of the external surface of the skull. They are covered with skin and communicate with the cranial cavity through a defect in the bony wall of the skull and appear in its median line either at the occiput or in the region of the glabella and root of the nose. Occasionally, the anomaly protrudes through the sphenoid or ethmoid bones into the nasal cavities.

Cephalocele is analogous to spina bifida (p. 1422). The most frequent form is the *hydrocephalocele,* which is covered with more or less atrophic skin, while the arachnoid, the bone and dura are absent. The last two merge at the base of the tumor. Within the arachnoid is a layer of brain substance surrounding a hollow space filled with cerebrospinal fluid, which communicates with the ventricles. The origin of the protrusion is traceable to incomplete closure of the cerebrospinal tube.

FIG. 744.— OCCIPITAL CEPHALOCELE.

The exact anatomical conditions are presented by Lyssenkow,[1] Muscatello,[2] Berger,[3] and others (see bibliography of Spina Bifida, p. 1432).

The *diagnosis* of cephalocele is made on the congenital presence of the tumor and its typical location. It is not infrequently associated with hydrocephalus.

In the few cases which survive after the first few days of life, the *treatment* consists in entire removal of the protuberance. The technic of the procedure is very similar to that described in connection with

spina bifida. Lyssenkow[1] closes the defect in the skull with a "free bone flap." However, if ablation of the protrusion succeeds, the defect closes as the child develops.

Congenital Hydrocephalus.— Congenital hydrocephalus, as distinguished from all other forms of hydrocephalus, presents a consistent and typical clinical picture. The condition consists in a continuous, progressive increase of the fluid of the ventricles of the brain — the lateral, the third, and the fourth, and at the same time a corresponding increase in size of the infantile skull, which, at that period of life, has not closed along the sutures or at the site of the fontanelles. The fluid may increase to 1000 or even 2000 c.cm. and does not show any chemical variation from the normal. The brain tissue is flattened as the result of pressure. The condition is clearly depicted in figure 745.

FIG. 745.— CONGENITAL HYDROCEPHALUS (Harlem Hospital case).

As the fluid collects, the pressure on the brain increases and the child dies, usually within the first year of life. At times the accumulation of fluid ceases spontaneously and then suddenly increases again. Complete cessation of the process occurs in a considerable number of instances, the ultimate fate of the patient being dependent upon the amount of pressure the brain has been subjected to.

The *treatment* of hydrocephalus is not satisfactory. *Repeated aspiration,* though not dangerous, is only palliative. Recovery has occurred in cases in which this has been done (thirty times at intervals of five to ten days). *Spinal puncture* is effective only when the foramen of Magendie is patent. *Operative measures* destined to establish a communication between the ventricles and the subcutaneous

tissues have met with varying success. The technic of Krause,[4] who uses a small silver tube for the purpose, has been followed by relief in a number of instances. The work of Kalisher[5] (who furnishes the literature) may also be studied to advantage.

Concussion of the Brain.— Concussion of the brain (commotio cerebri), like that of the spinal cord (p. 1448), has been made the object of prolonged investigation for a time, with apparently widely divergent conclusions. The details of this need not be entered into here. From the ruins of the contest, the facts remain that injury of the brain (and skull) is attended with varying degrees of disturbances of consciousness without the least evidence of an anatomical lesion of the brain, and that severe trauma of the brain with contusion, laceration, and even extensive loss of substance, may not be attended with modification of consciousness. Therefore, one is driven to the inevitable conclusion that THERE IS SUCH A THING AS PURE CEREBRAL CONCUSSION and that it represents a clinical entity. Its association with cerebral contusion and laceration goes without saying, and its clinical differentiation in this event is not possible at first. Yet its existence must be acknowledged, more especially as it has been shown that pure concussion of the brain may result in death although autopsy may not reveal evidence of an anatomical lesion (see Kocher,[6] Polis,[7] Bouillard,[8] Roncali,[9] Miles,[10] Levi[11]). V. Bergmann[12] says "The disappearance of consciousness while the vital forces persist indicates an arrest of the function of the cortex of the cerebrum. After a fall upon the head the patient is incapable of conscious effort, and yet, during this night of the senses the medulla oblongata continues to functionate, a picture which can be produced only by the disturbance of the function of the brain as a whole; this is the determining factor which justifies the conception that there is such a thing as pure cerebral concussion." Experimentally it is possible to produce the picture of commotion without producing a discernible lesion of the brain (Kocher[6]). Just what the pathogenesis of cerebral concussion is is not susceptible of demonstration for obvious reasons, and one must be content with the belief that it is the outcome of the transient effect of trauma upon the brain, and this may be circulatory (Fischer[13]).

Injury of the skull causes changes in its configuration in three ways, (1) in the sense of *concussion* (*commotio cerebri*), (2) through the collection of endocranial extravasation which occupies space in the cranial cavity and interferes with the circulation (*compressio cerebri*),

(3) by the crushing and laceration of the cranial contents (*contusio cerebri*).

All of these are due to trauma; they may exist alone or may be associated with one another. A lesion may begin as concussion and merge into compression, or contusion may occur primarily. Concussion and compression are general disturbances of the brain; contusion is localized and is fatal when a vital center (medulla) is involved. Concussion and compression are attended with a constant clinical picture; contusion is attended with spasm, paralysis and disturbances of the special senses (speech, sight and hearing) and of respiration and heart's action.

The *clinical picture of cerebral concussion* is expressed in disturbances of consciousness, which may be *mild* or *severe*. The *mild forms* are attended with dizziness, flashes before the eyes, and buzzing in the ears; consciousness is entirely lost; there is complete exhaustion, the knees give way and the arms are relaxed. The face is pale, the eyes fixed and expressionless and the eyelids closed. Respiration is shallow, the pulse feeble. This condition is of short duration. The pulse improves; this is followed by several deep respirations (vomiting is common); the patient opens his eyes, stretches his arms and gets up. On attempting to stand he is unsteady, grasps his head, complains of pain in it and at the site of injury, of buzzing in the ears, soreness of the entire body and of great lassitude. Gradually all the symptoms disappear.

In the severe cases, the patient collapses at the moment of injury and remains absolutely motionless, as if in deep sleep. He cannot be aroused, reacts to no external stimulus, nor attempts any defensive motion. In all cases the pupil reacts to light, and if water is placed in the mouth, the patient swallows. The surface of the body is cool, the face is shrunken and pale. Respiration is shallow, the pulse small and irregular. Feces and urine may be expelled involuntarily. This condition may persist for hours and even days. Finally, respiration becomes deeper, heart and pulse stronger and fuller, the body regains its normal warmth, and, at the same time, consciousness and the power of motion return. The senses are unimpaired and questions are answered clearly. As a rule, the above stage of *depression* is followed by a period of *exaltation*. The pulse increases in frequency and becomes hard, the surface temperature is increased, the face is red, the pupils contracted, the eyes bright. The duration of this stage is variable. *If the symptoms continue for several days, the diagnosis of*

pure concussion is excluded. The longer the state of unconsciousness continues, the more may the presence of intracranial injury be assumed. Only in cases in which dangerous symptoms subside rapidly, can the occurrence of extravasation and consequent intracranial pressure be excluded; while conversely, in those cases in which the manifestations increase in severity from hour to hour — the pulse becoming slower, coma more deep, and respiration intermittent — it is fair to assume that further disturbances are present and that these are the outcome of continuously increasing intracranial extravasation.

The *treatment* of cerebral concussion is that of shock (p. 945). In the stage of exaltation morphia may be given subcutaneously. Ritter[14] advises stimulating return of cerebral function by active and passive motion of the limbs, dictation of a pleasant theme and mental occupation — a sort of educational training.

Compression of the Brain.— Compression of the brain is a term applied to a symptom complex of great clinical importance. It enables the surgeon to recognize laceration of the middle meningeal artery, the presence of a brain tumor, and is of assistance in diagnosticating abscess of the brain and a number of acute and inflammatory complications of brain injuries. The COMPRESSION IS THE OUTCOME OF LIMITING, IMPEDING AND RETARDING THE CIRCULATION OF FRESHLY OXYGENATED BLOOD THROUGH THE BRAIN BY LESSENING OF THE SPACE OF THE CRANIAL CAVITY (Geigel[15]).

Slowing of the circulation in the brain immediately lessens the functional activity. This varies in degree in the several portions of the brain. As the circulation becomes gradually slower, the centers are affected in sequence as follows, the cortex, the corona radiata, the gray matter of the cord, the pons, and, finally, the medulla oblongata. Assuming this to be the case, it follows that when certain centers are paralyzed, others only begin to be affected. Therefore, unconsciousness is combined with convulsions.

The *clinical picture of cerebral compression* is presented as follows: In uncomplicated cases the causative factor (trauma, sudden effusion of blood) is followed by a certain interval before the symptoms appear — the so-called *free interval.* This distinguishes concussion and contusion from pure compression. Attention is called to a constant early symptom of cerebral compression by Bordley and Cushing,[16] who state that the visual field for blue is smaller than it is for red, and for white is normal. After the pressure is relieved normal conditions are reëstablished.

OF ALL THE FUNCTIONS, THAT OF CONSCIOUSNESS IS MOST RAPIDLY LOST and does not return until the circulation is fully reëstablished. As a rule, however, a case of pure compression is rarely encountered; most of them are associated with concussion and contusion of the cranial contents, so that the study of compression by itself is of necessity based on experimental observations. These show that the PRESSURE SYMPTOMS DEVELOP IN REGULAR SEQUENCE AND, IN A GENERAL WAY, THE SYMPTOMS APPEAR IN ACCORD WITH THE DEGREE OF PRESSURE.

The onset is attended with restlessness, groaning, nausea and vomiting, followed by unconsciousness, stupor and coma. SLOWING OF THE PULSE OCCURS SIMULTANEOUSLY WITH STUPOR, AND IS ONE OF THE MOST CONSTANT AND CLEARLY MARKED SYMPTOMS OF CEREBRAL COMPRESSION. When the pressure exceeds a certain degree the pulse becomes rapid. In the transitional stage the heretofore regular, full pulse becomes small and intermittent. This is ascribed to irritation of the *vagus* in the first instance and its paralysis in the second. The *vasomotor center* presents a similar condition of affairs; thus the blood pressure at first rises and later falls (v. Bergmann[17]). *Respiration,* primarily superficial, soon becomes *slow and often stertorous.* As is the case with the heart, when the compression of the brain is carried beyond a certain point, respiration becomes again irregular, with long intervals of absence of respiratory effort, until these cease entirely.

The *symptomatology of cerebral compression* may be divided into *two stages,* that of irritation attended with cephalalgia, vomiting, restlessness, congestive redness of the face, contracted pupil, development of choked disk, increased blood pressure and slowing of the pulse; and *paralysis,* in which unconsciousness merges into stupor and coma, respiration becomes stertorous and intermittent, the pulse rate greatly increases, and the urine and feces are expelled involuntarily. Presently, the respiration becomes of the Cheyne-Stokes variety and soon ceases entirely, while the heart continues its laborious activity for a few moments longer. The severity of the pathological changes is indicated by the ophthalmoscopic examination, which reveals swelling of the optic nerve, i. e., *choked disk.* The precise pathology of cerebral compression is presented in detail by Tilmann,[18] who reviews the work of a number of other observers. There can be no doubt that increased blood pressure in the arteries of the brain is conveyed to the cerebrospinal fluid, which is caused to flow into the spinal canal, thus affording, as far as possible, the necessary space for the increased quantity of blood in the cranial cavity. As long as this balance is

maintained cerebral compression does not occur; beyond certain physiological limitations, however, the clinical picture indicative of brain pressure begins to develop. This is equally applicable to lessening of the space in the cranial cavity from other causes which may be *acute or chronic.*

Acute cerebral compression occurs in connection with depressed fractures and extracerebral hemorrhage and acute inflammatory processes (meningitis purulenta), acute brain abscess, epidemic cerebral meningitis, and meningitis serosa.

Chronic cerebral compression develops from congenital and acquired chronic hydrocephalus, chronic brain abscess, cysts and parasites of the brain, and from premature closure of the sutures and fontanelles of the skull.

Tuberculous meningitis occupies a place between acute and chronic compression.

In a general way, it may be said that the more sudden the compression, the more acute are the symptoms. ACUTE COMPRESSION is practically limited to ENDOCRANIAL TRAUMATIC EXTRAVASATION OF BLOOD AND EXTENSIVE DEPRESSED FRACTURES OF THE SKULL. The latter are more important in connection with localized pressure than with general compression. To these primary causative factors must be added the effect of the *accumulation of the products of inflammation* in the form of acute or chronic abscesses.

The *course and outcome of cerebral compression* are determined by the character of the causative factor. Much depends upon whether the compressive agent is subject to modification in its relationship to the capacity of the cranial cavity, i. e., whether the encroachment is progressive or whether it permits of resumption of the normal. In bone pressure compression progresses, unless relieved by operative measures. An extravasation of blood may be arrested by spontaneous closure of the vessel.

Purulent exudates go relentlessly on. IT IS, THEREFORE, OF THE GREATEST IMPORTANCE TO DETERMINE WHETHER THE PRESSURE SYMPTOMS ARE INCREASING OR DECREASING. Next in importance is THE LENGTH OF TIME THE COMPRESSION HAS EXISTED. Even severe pressure of *short duration* is soon recovered from. *Persistence* of the pressure is no less dangerous than its degree, on the ground that slowing of the circulation is followed by transudation of the fluid constituents of the blood and the development of cerebral *edema.* In this event, regression of the symptoms of compression is very slow and may never occur.

The *treatment of cerebral compression is aimed at its cause.* This means the operative elevation of the depressed bone and the removal of extravasated blood, the inflammatory exudate, the tumor and the excessive amount of cerebrospinal fluid. THIS IS ATTAINED BY TWO METHODS, FIRST, BY PUNCTURING OR DRAINING THE VENTRICLES OR THE SUBARACHNOID SPACES AT THE LUMBAR REGION OF THE SPINE, AND SECOND, BY OPENING THE SKULL.

Reduction in the quantity of the cerebrospinal fluid is indicated in acute hydrocephalus of tuberculous meningitis, acute serous meningitis and chronic acquired hydrocephalus.

The methods of reducing the quantity of cerebrospinal fluid are:

1. Lumbar puncture and lumbar drainage.
2. Puncture of the ventricle, cerebral puncture and ventricular drainage.
3. Decompressive trephining.

(1) For *lumbar puncture* see Part IX, chapter i, especially the bibliography.

(2) Puncture of the lateral ventricle may be accomplished from in front (v. Bergmann[17]) or through the parietal bone (Keen[19]). Permanent drainage has been attained by Keen,[19] Krause,[4] and others by inserting glass or metal cannulas through small trephine openings into the ventricle and establishing communication with the subaponeurotic space (see also Payr,[20] Fowler[21]).

(3) *Decompressive trephining* is employed when the compressing agent cannot be removed, i. e., not localized and inoperable tumors. Its great *disadvantage* lies in the sequential prolapse of the brain (hernia cerebri) and the trauma to the brain tissue. The latter argues for the selection of a site over an unimportant sector of the brain.

Injury of the Intracranial Vessels.— Injury of the intracranial vessels is of the greatest practical importance in connection with the *middle meningeal artery.* The artery is injured by penetrating instruments and by projectiles. In the majority of cases, however, blunt force produces more or less extensive bone injury, as the result of which the vessel is torn. It is most frequently lacerated as the bone is broken. This is more likely to occur when the artery lies in a canal than in a groove. The artery has been torn without the bone being fractured, i. e., as the result of temporary deformation of the skull. Injury to the vessel by *contre coup* is of especial interest and its possibility must be borne in mind in the diagnosis. Hovanian[22] has collected thirteen

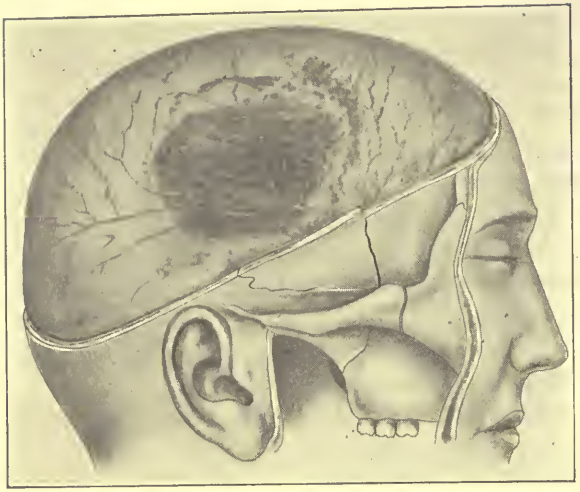

FIG. 746.— *A*, COMPOUND FRACTURE OF CRANIUM WITH DEPRESSION.
Fracture of bones of face; extradural clot from rupture of middle meningeal artery.

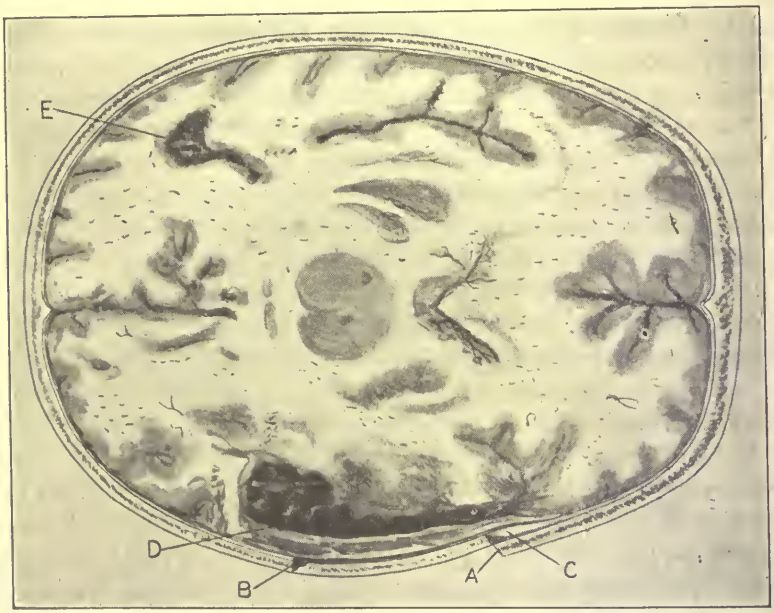

FIG. 746.— *B*, HORIZONTAL SECTION OF *A*.
Depression of bone A-B; Extradural clot C; Laceration of brain substance with intracerebral clot D; same condition (E) produced by *contra coup*. Punctate hemorrhages and minute lacerations at numerous points, characteristic of contusion of the brain.

cases of this sort. The mechanism of laceration of the artery is encompassed in the "mechanics of fracture of the skull" (p. 1489).

The immediate effect of laceration of the artery is bleeding which, as it cannot escape from the cranial cavity, is followed by the accumulation of blood (hematoma) within it, unless the injury occurs in connection with a compound fracture. The result of the formation of the hematoma is *progressive compression of the brain.* In most instances the extravasation of the blood is extradural. Occasionally, when the dura is torn the blood is effused subdurally. As the extradural extravasation progresses the dura is elevated from the bone (*zone décollable* of Marchant[23]). Spontaneous arrest of bleeding, the result of increased intracranial pressure, occurs when about 200 gms. of effused blood has collected (Krönlein[24]); clinical manifestations develop when 60 gms. of blood have been effused (v. Bergmann[12]). The hematoma is usually situated in the middle fossa of the cranium. The posterior branch of the vessel is rarely torn (Krönlein[24]). The morphology of the effused blood has been studied by Wiesmann.[25]

The *clinical manifestations* of intracranial hematoma are expressed in the *typical symptom complex of increasing cerebral compression,* usually preceded by a *free interval of several hours.* The picture of general compression is not infrequently preceded by the gradual development of *contractile hemiplegia,* especially when the hematoma is still circumscribed. The claim that the *pupil* on the injured side is always dilated and that on the other contracted has not been substantiated.

The *diagnosis,* in practice, is not without its difficulties. These are embraced in the fact that most cases at first display the symptoms of concussion or contusion, which may be so severe that there is not time for them to subside before the stage of compression begins to become manifest. This is especially true, as focal symptoms invariably occur late in the symptomatology of head injuries and, not infrequently, slowing of the pulse, due to irritation of the vagus center, occurs early. When a head injury occurs during alcoholic intoxication, the picture is still more obscure, to which may be added the difficulties attendant upon a sudden apoplexy which causes the patient to fall and strike the head. In doubtful cases, brain puncture gives valuable information. When a fracture is present, much reliance may be placed on the radiographic findings (p. 1494).

The *prognosis,* when the pressure is not relieved, is uniformly unfavorable.

The task of TREATMENT consists in RELIEF OF THE CEREBRAL COMPRESSION BY REMOVAL OF THE HEMATOMA AND PREVENTION OF FARTHER ACCUMULATION OF BLOOD. In OPEN INJURIES this is readily accomplished by opening the wound in the skull; wiping out the clot, and tying the bleeding point or points.

In *closed injuries* the skull must be opened. This should be accomplished over the main trunk of the middle meningeal artery. As it is rarely certain which of the branches of the artery is torn, the osteoplastic trephining of Krause[4] is largely practiced for the purpose. The technic is the same as that used for exposure of the ganglion of Gasser (p. 1626). The exposure achieved in this way renders the artery readily accessible, permits of complete removal of the hematoma, of ligature of the torn vessel, and makes satisfactory repair of the skull possible. The division of the main trunk of the artery into its two primary branches corresponds to a point 4 cm. behind the zygomatic process of the frontal bone on a line drawn from the supra-orbital ridge backward and parallel to the horizontal base line of the skull (Krönlein[24]).

When the extravasated blood is removed the clinical picture *immediately changes,* so that not infrequently return to normal occurs simultaneously with recovery from narcosis. Few surgical efforts at relief are crowned with a more brilliant outcome than is the successful achievement of the abolition of cerebral compression in cases of this sort.

Injuries of the Cranial Sinuses.— Injuries of the cranial sinuses of surgical importance are those involving the longitudinal, the lateral, and the junction of the two — the torcular Herophili. They occur most frequently in connection with fracture of the vault of the skull, in which the bony structure breaks and tears the sinus or, more commonly, a splinter of the internal table perforates one of the sinuses. A similar condition of affairs occurs in connection with punctured or gunshot wounds, in which either a splinter of bone or the projectile traumatizes a sinus. It is not improbable that a sinus may be torn when blunt force deforms the skull without the occurrence of fracture; to these belong a certain number of the cases of intracranial hemorrhage of birth (see Wharton[26]).

In open injuries of a sinus the *symptoms* are self evident; in those in which the cranium is intact the picture of cerebral compression is presented. *Diagnosis* is possible only in open injuries.

The *surgical treatment,* i. e., the arrest of bleeding, is also limited

to the open injuries and consists in exposure and tamponade of the sinus. Double ligature of a completely torn sinus is not impracticable. Trephining for the relief of cerebral compression in the closed cases would have to be based on localization of the hematoma by brain puncture and on a consideration of the manner in which the injury was received. The treatment of sinus perieranii is taken up on page 1484.

Injuries of the vessels of the pia (subdural hemorrhage) are attended with bleeding into the meshes of the pia mater. The extravasations are widely varied in extent, ranging from a minute ecchymosis to a hematoma covering an entire cerebral hemisphere. The condition not infrequently occurs in *the newborn* in connection with forceps deliveries. In these instances the clinical picture closely resembles the asphyxia of the newborn. Debraigne[27] advises the employment of lumbar puncture, which clears up the diagnosis. Cushing[28] has employed bilateral subtemporal decompression for relief of the pressure. Of nine cases, four recovered.

Traumatic subdural hematoma is so often associated with contusion and laceration of the brain that the presentation of a distinctive clinical picture is not to be expected. The symptom complex is very similar to that of extradural hemorrhage. Henschen[29] divides the condition into *diffuse and circumscribed hematomata.* The latter may be situated anywhere in the brain. Those at the cerebellum and medulla are rapidly fatal.

Perhaps the only *distinctive diagnostic phenomenon* is that emphasized by Oppenheim,[30] namely, that the evidence of cerebral involvement does not become manifest until the fifth or seventh day after the injury. The lesion may be attended with the presence of blood in the fluid obtained by lumbar puncture. In a general way the *prognosis* of subdural hemorrhage is better than that of the extradural; the former is more likely to be absorbed.

The *treatment* is indicated by the gradual domination of the clinical picture of cerebral compression. It consists in preliminary cerebral puncture and, when the site of the bleeding is determined, in trephining and removing the clot. Brion[31] calls attention to the fact that removal of a certain portion of the extravasated blood is followed by accelerated absorption of the remainder.

Traumatic arteriovenous aneurism of the internal carotid artery (sinus cavernosis) results from fracture of the base of the skull and is caused by penetrating and gunshot wounds of the vessel in this situation.

In fully developed cases the symptoms are characteristic. They consist in a marked *exophthalmos,* in which the eyeball distinctly *pulsates,* and a clearly defined *bruit* is present. The eyelids are edematous and the veins about the orbital cavity are dilated. Examination of the fundus reveals *choked disk* and the presence of dilated veins. The various motor nerves of the muscles of the eyeball are progressively paralyzed and the fifth nerve is often affected. The patient complains of headache and is constantly conscious of buzzing and roaring in the ears. Compression of the carotid in the neck excludes the symptoms, which return when the pressure is released.

The *treatment* is not very satisfactory. Digital compression has been employed with lessening of the symptoms. Ligature of the common carotid is justified. In this connection the monograph of Becker[32] is instructive.

The operative treatment of apoplexia sanguinea has long fascinated the imagination of the surgeon. In 1890, Horsley[33] proposed to tie the carotid in cases of this sort. Chipault,[34] on theoretical grounds, suggests that in individuals who have already had an attack of apoplexy, the carotid may be tied as a preventive measure, and in persons who have a mild primary attack, the clot may be removed and the cavity tamponed. Marion[35] regards the first proposition as objectionable, on the ground that the operation has no influence on the causative arterial disease, and fears that the identification of the focus is too uncertain to justify the second. More recently, Franke[36] and Milligan[37] have reported cases in which enucleation of the clot has been followed by recovery. Both surgeons operated during rather severe symptoms of compression. On the other hand, Marion[35] considers the operation justified only when localization of the lesion is possible.

Injuries of the cranial nerves in their course in the skull occur in connection with severe trauma of the cranium as a whole, usually the base. However, at times, a nerve in this situation is directly injured in perforating and gunshot wounds. As a rule, paralysis of a cranial nerve occurs at the time the trauma is inflicted, i. e., by crushing or laceration by a splinter of bone; occasionally, late paralysis results from pressure of reparative callus. In primary injuries the prognosis is good, the nerve usually regenerating. When the paralysis occurs late, return of function does not, as a rule, occur. The clinical picture is that of paralysis of the part supplied by the injured nerve (see Surgery of the Nerves, p. 574). Special presentations on the subject

are submitted by v. Bruns,[38] Stephanoff,[39] Bonner,[40] Cantonnet,[41] Ferron,[42] Clairmont,[43] and Honusa.[44]

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CHAPTER III

CONTUSIONS AND WOUNDS OF THE BRAIN

Excluding the rare, *direct* invasion of the cranial contents through the sphenoidal fissure, injuries of the brain take place *indirectly* through the medium of the bones of the cranium. This is brought about by external violence, the outcome of either a change of conformation of the skull (and its reassumption of the normal) or of a fracture or wound of the bones. However, even punctured and lacerated wounds are attended with more or less contusion of the brain. The latter is in accord with the hydrodynamic laws and their influence upon the soft consistency of the brain substance, which easily transmits force in all directions, and, therefore, also in a direction perpendicular to that of the inflicted violence. For practical purposes, traumatic lesions of the brain may be divided into *contusion of the brain,* in which the solution of continuity does not communicate with the air, and *wounds of the brain,* i. e., injuries occcurring in connection with open wounds in the covering of the brain and involving the brain itself, the path of the trauma communicating with the open air.

Contusions of the Brain.— Contusions of the brain present wide differences as regards extent, number and location. They may consist of small punctuate areas of contusion, or may be the size of a cherry or a goose egg. An entire lobe may be crushed.

The localization of brain contusions is extremely varied. At the first glance they appear to be subject to no rule, yet are found to occur in a certain regular order, if considered not by themselves, but in connection with the fractures of the skull which so frequently accompany them. THE MECHANISM OF THE PRODUCTION OF CONTUSIONS IS THE SAME AS THAT CONCERNED IN THE PRODUCTION OF INJURIES OF THE SKULL AND THE BRAIN AT THE SAME TIME AND MAY BE FORMULATED AS FOLLOWS:

1. In *bursting fractures* of the skull the accompanying contusions of the brain generally lie in the direction of the inflicting violence, being usually more severe at the poles of the line of force, i. e., at those portions of the brain which receive the direct impact at the time

the spheroid becomes flattened. Thus, in *longitudinal fractures* of the base the frontal lobe of the cerebrum and the cerebellum are contused; in *transverse fractures,* both temporal lobes; in *oblique fractures,* the frontal lobe of one hemisphere and the parietal lobe of the other are contused. Deviation from this general law is often found, owing to the peculiarities of the cranial spheroid and of the brain substance itself (Krönlein[1]).

2. In *localized comminuted fractures* of the skull the principal contusions appear immediately beneath the site of injury.

3. The occurrence of *multiple foci of contusions* is explainable on the ground of the semifluid consistence of the brain and the abundance of its blood and cerebrospinal fluid. According to hydrodynamic laws, any violence affecting this mass at one point can be transmitted in directions other than the line of force, producing solution of continuity of tissue. Relatively, the walls of the ventricles appear to be most frequently the site of contusion when the direction of the violence coincides with the major axis of the cavities, i. e., in connection with longitudinal fractures.

4. Where there are *deep lacerations of brain substance,* they are to be considered the result of bursting fractures in the direction of the inflicting violence.

Laceration of the meninges may occur in injuries of the skull independently of contusion. In these instances the solution of continuity of the dura or pia is due to the same force that produces a simultaneous contusion at another portion of the brain.

The *course of contusions of the brain* which communicate with the air is similar to that of wounds in general, and is of course most seriously influenced by infection (p. 1526). In those that do not communicate with the air the changes are those common to subcutaneous contusions in general. In the brain the connective tissue elements of the pia and the vessels play almost the only part, while the neuroglia has an insignificant one. Regeneration of nerve cells, and probably of nerve fibers, does not occur. This much is certain, EXTENSIVE TRAUMATIC DEFECTS OF THE BRAIN ARE CLOSED BY THE DEVELOPMENT OF SCAR TISSUE (Ziegler,[2] Tschistowitsch[3]). This scar may be solid and firm or may show a cystoid structure, the cavities of which are filled with a yellow fluid (cholesterin).

Brief allusion may be made to *rupture of the brain occurring in children,* the result of injury. The condition is characterized by a distinct tear in the brain extending into the ventricle. This heals and

then forms a cyst of, at times, considerable magnitude (*porencephalia traumatica*). These injuries and the sequential cyst formation are responsible for so-called *meningocele spuria,* or *cephalohydrocele traumatica,* which bears considerable resemblance to congenital cephalocele described on page 1502 (Rahm[4]).

The *processes of repair following contusion of the brain* have a bearing on the present day conception as to the justification of operative efforts destined to afford relief from the unfortunate sequelae consequent to this class of injury. Aside from the protracted process of repair which results in scar formation in accord with the degree of tissue destruction, there are cases in which, during the course of years, a degeneration of the nervous elements takes place which extends far beyond the limits of the original injury. This *encephalomalacia is* ascribable to a progressive degeneration of the small blood vessels, brought about by circulatory disturbances in the vicinity of the area of contusion. The process may (after a prolonged latent period) extend over several lobes of the brain and destroy the host (v. Monakow[5]). The influence that these histological changes have upon various *psychoses, cortical epilepsy, cerebral tubercle, glioma, sarcoma,* etc., cannot be disregarded (Küttner[6]).

Wounds of the Brain.— Wounds of the brain may be classified as *contused, punctured,* and *lacerated.* In distinction from those just discussed, they possess one common feature, namely, that the causative violence has divided all the coverings of the brain, so that there is a path from the external surface of the body to the central organ of sufficient size to allow the escape of destroyed brain substance on the one hand, and the entrance of air and infectious germs on the other. This exposure of the brain lesion is a factor of such great importance that a distinction between contusion and wounds of the brain seems justifiable.

Wounds of the brain occur as the result of violence from blunt or sharp objects. Both mechanisms may occur at the same time. In fact, as a result of the involvement of the cranial bones, the provocative force may be entirely altered in its transmission from without inward, so that blunt force may lead to a brain injury which possesses the characteristics of a punctured or incised wound, while violence from a sharp object may lead to a contused wound. The nature of these forces and their effect are in accord with the mechanism of the production of fractures of the skull (p. 1489).

The *course of a wound of the brain* does not depend so much upon whether it bears the character of a punctured or lacerated wound, as

upon whether infection has or has not taken place. If infection does not occur, the subsequent course of wounds of the brain is the same as that of contusions of the brain. The course of infected wounds is taken up below.

Contused wounds of the brain occur in connection with compound fractures of the skull, especially those caused by projectiles. In the latter instance the trauma to the brain tissue is likely to be great and the sequelae, previously discussed, most frequently develop. This is especially important in connection with gunshot wounds of the brain produced in civil life by small arms (Flobert rifles, etc.).

Punctured wounds are of import because of the danger of infection and the liability that portions of the causative instrument may be left *in situ*. When infection does not occur, it frequently happens that objects of this sort become encysted and do not give rise to any disturbances.

Lacerated wounds of the brain do not present any determining regional peculiarity. They are often produced by sabers, cleavers, heavy knives, etc., and are less likely to be infected than the contused and punctured. However, as already stated, the causative force often produces contusion of the adjacent portions of the brain tissue, especially when the bone is splintered.

Infection of wounds of the brain is the outcome of the entrance of pyogenic organisms of a certain virulence into a field receptive for colonization. Unfortunately, *wounds of the brain are particularly susceptible to infection*. This is shown by the fact that what appears to be a simple contusion of the brain is often followed by the development of infection. In such cases pyogenic organisms are conveyed to the site of the lesion through the circulation; suppuration is here a metastatic process. However, this is exceptional, and as a rule there is a more direct invasion of the excitants at the site of the injury. If the traumatized area is thus invaded, suppurative inflammation develops. This entire process runs the anatomical and clinical course of *encephalomeningitis*. If the inflammation remains localized and the pus readily escapes through the existing wound in the bone and soft parts, repair may still take place by granulation and cicatrization. If, however, the pus does not find an avenue of egress and is retained in the injured area, an *acute traumatic cortical abscess* is formed. Frequently, however, purulent inflammation is not restricted to the injured area, but, from the very beginning, exhibits a decidedly progressive character, spreading rapidly in the loosely meshed connec-

tive tissue of the arachnoid. The pial tissues and the subarachnoid spaces are soon filled with exudate extending along the vessels and invading the cerebral cortex. Spreading inflammation of this sort rapidly ends the life of the host. The organisms concerned in this process are usually the *streptococcus pyogenes* and the various *pyogenic staphylococci.*

The *symptoms of brain injuries* are usually those indicating the destruction of *localized areas of cerebral tissue* and the *diagnosis* rests upon a sufficient knowledge of the localization of the physiological functions of the brain, as far as they are determined by experimental and clinical observations. While the history of the case and the location of the injury should be, and are, helpful in diagnosticating the site of a traumatic brain lesion, a conclusion in this connection is often rendered difficult by the following factors:

1. Very frequently contusions and wounds of the brain are associated with manifestations of concussion and compression which completely obscure the focal symptoms.

2. In many instances there is more than one traumatized area in the brain.

3. The areas of injury, particularly in contusions, are often very extensive and are combined with intrameningeal hemorrhage, hemorrhage into the cortex and into the ventricles.

4. A number of localized injuries involve portions of the brain and do not give rise to symptoms.

5. While, until now, only symptoms of primary localized injury produced by mechanical causes have been considered, it is necessary to take into account that frequently symptoms of infection rapidly appear. These are the expression of purulent meningitis and encephalitis and indicate the site of the lesion. More frequently, however, as a result of these progressive changes, the nutrition and circulation in the entire brain are so changed that a topical diagnosis is impossible.

Cerebral Localization (After v. Monakow[5]).—The *motor zone* encompasses the cerebral cortex of the *anterior central convolution,* a part of the *posterior central convolution,* the *gyrus centralis,* the *lobulus paracentralis,* the *operculum* and the *root of the third frontal convolution.*

The *central convolutions contain three cortical zones:*

1. The leg region, in the upper one fourth of both central convolutions (including the paracentral lobule).

2. The arm region, in the middle two fourths.

3. The head region, in the lower one fourth (including the operculum, central fissure, and the root of the third frontal convolution).

Each of these three regions include the various foci for excitation of *definite groups of muscles.*

FIG. 747.— CEREBRAL LOCALIZATION.

The posterior central convolution is probably concerned in sensation. On the other hand, there is no doubt that a portion of the frontal lobe in front of the anterior central convolution, certain sectors of the occipital lobe (motion of the eyeballs), and the temporosphenoidal lobe (movements of the ear) are motor.

For *contusions and wounds within the motor zone,* the following laws may be established for diagnostic purposes:

1. Cortical lesions alone are rare. As a rule, the injury extends to the white substance beneath.
2. Very small cortical lesions may occur without symptoms, if an unimportant area is involved.
3. *Per contra,* a slight lesion may produce a characteristic symptom complex, when located in an important focal area.
4. Total destruction of both central convolutions always causes *hemiplegia on the opposite side of the body.*
5. *Muscle sense* is impaired when the parietal lobe is injured; *tactile sense* without disturbance of sensation occurs in lesions limited to the posterior central convolution.
6. *Partial destruction* of both central convolutions is common and characterized by *monoplegias and monospasms.*

These *monoplegias* are either *pure,* i. e., paralysis of an isolated part of the body (leg, arm, head), or *associated,* i. e., paralysis of greater or lesser areas of two regions simultaneously; for instance, *arm and leg* (*brachiocrural monoplegia*) or *arm and head* (*brachiofacial monoplegia*); never, however, leg and head without the arm, because the head and the leg are separated by the arm center.

Among the *pure* monoplegias, that of the *leg* is observed in lesions of the *upper one fourth of the anterior* and also of the *posterior central convolution* or of the *paracentral lobule.* In mechanical irritation (splinters of bone), monoplegia is preceded by a period in which the leg is contractured and may exhibit spasm. A cortical lesion of this sort may give origin to *jacksonian epilepsy.*

Monoplegia of the arm is the result of a lesion of the *middle half of the central convolutions, including the cortex of the contiguous sulci.* Traumatic paralysis may be preceded by clonic spasm, and *jacksonian epilepsy* may take its origin from this location.

Isolated cortical facial paralysis does not occur. Lesions in the head region (facial and hypoglossal regions) are attended with associated *faciobrachial* or *faciolingual monoplegia.* In so-called facial monoplegia, the lower facial muscles are usually affected, the upper escaping. In *faciobrachial monoplegia* the muscles of the tongue and forearm are usually affected.

The *parietal convolutions* are believed to be concerned in the higher psychic functions, and the center of this region, in *muscle sense.* Lesions in this situation may be responsible for volitional ataxia and for sensory *jacksonian epilepsy.*

Lesions of the *occipital convolutions* are attended with *hemi-anopsia* (lateral). This is due to the decussation of the optic nerve fibers in

the chiasm. The region of the calcarine fissure is especially concerned in this connection. Complete *hemi-anopsia* (hemi-amblyopia, hemi-achromatopsis) occurs in lesions of both occipital lobes. Cortical lesions of these lobes are at times attended with *optic aphasia.*

The functions of the *frontal convolutions,* aside from the posterior ends of the first and second (motor) and the posterior half of the third — which is concerned in *speech* — are not clearly understood. They would seem to be concerned in the so-called intellect. In tumors of the frontal lobes, a form of ataxia (*frontal ataxia*) resembling that attendant upon lesions of the cerebellum has been observed (Jastrowitz, Witzelsucht, L. Bruns, Liepmann, Hartmann, see v. Monakow[5]).

The *speech center* encompasses the cortex of the region around the Sylvian fossa, i. e., the *posterior half of the third frontal* (Broca's) *convolution, the entire island of Reil, the first temporal convolution, including the cortex of the entire Sylvian fissure to the supramarginal gyrus.* Lesions in this region produce the disturbances designated by the term *aphasia.* In right handed persons, the left side of the region stated, and in left handed persons, the right side functionates in this connection.

Motor aphasia consists in an inability to express ordinary language or to repeat spoken language. This is due to a lesion of the (left) third frontal convolution and is often associated with *agraphia* (inability to write).

Sensory aphasia (word deafness) consists in inability to understand spoken language. This is caused by a lesion in the (left) first temporal convolution and is often associated with *alexia* (inability to understand printed words), rarely with *agraphia,* although, paraphasia is present.

Complete aphasia occurs when motor and sensory aphasia coëxist. It is easy to understand that a severe lesion of the third frontal convolution would extend to the contiguous structures of the motor region and that the head center would be likely to be involved. As a matter of fact, motor aphasia is often attended with *faciobrachial and faciolingual monoplegia and even with hemiplegia.* When lesions of this sort are irritative in character, *jacksonian epilepsy* is often present.

In lesions of the *upper temporal convolution* (including the gyrus supramarginalis) sensory aphasia may be associated with *disturbances of sensation,* especially of *muscle sense* on the opposite side of the body when the parietal lobe is involved; or it is combined with *hemi-anopsia* when the deeply located optic fibers (*en route*) are involved.

In the *insula* itself small lesions may not give rise to any disturbances, while larger ones produce motor or sensory aphasia, depending on the center to which they are nearest.

Lesions in the *internal capsule* are attended with symptoms in accord with the extent of the destruction of fibers, varying from *complete to partial hemiplegia and hemi-anesthesia.* Pure isolated monoplegia does not occur.

Lesions of the *corpus striatum* and the *lenticular nucleus* only produce symptoms when the internal capsule is involved.

Lesions of the *optic thalamus* are attended with *hemi-anopsia,* provided the *external geniculate* body and the pulvinae are involved.

Lesions of the *corpora quadrigemina* are attended with disturbances of vision (slight), a fixed dilated pupil, interference with the ocular motility, and ataxia (of the cerebellar type).

Small lesions of the *pons* do not produce symptoms. In the majority of cases, however, typical pons symptoms are presented. These consist in *alternating paralysis,* for example, left hemiplegia of the extremities, slight involvement of the face on that side, but complete paralysis of the right side of the face. A single nerve arising at the pons — for example, abducens, facial, hypoglossal — may be paralyzed.

The principal symptom of a *cerebellar lesion* is so-called cerebellar ataxia (le marche d'ivresse), very similar to the gait of alcoholic intoxication.

Involvement of the *medulla oblongata* is characterized by a complete or incomplete paraplegia or hemiplegia of the extremities, also anarthria, dysphagia, aphonia, singultus, disturbances of respiration, and finally glycosuria, polyuria and albuminuria, the last three indicating a lesion of the fourth ventricle.

The *treatment of recent injuries of the brain,* in cases in which localization of the lesion is impossible, is, of necessity, limited to the care of the injury of the skull. It is a comforting thought that, as a rule, wounds of the brain do best when not tampered with.

The fact that a brain injury is susceptible of localization does not of itself justify intervention. Indeed, as Krönlein[1] very properly asks, "What can intervention accomplish? The repair of a brain injury is accomplished, as far as is possible, without our help; and more than that is not to be hoped for."

Only two conditions appeal for surgical aid, and these are questionable, the presence of a foreign body, such as bone splinters, sub-

stances like portions of steel weapons, projectiles, etc., and the prevention of *septic infection.*

As regards a foreign body, determination of its presence may be fraught with great difficulties. This is not so difficult when the mechanism of the injury justifies a conclusion in this connection. To this class belong gunshot wounds with ports of entrance and exit; stab wounds in which the corpus delicti, a knife blade, nail or hook remains embedded; and open splintered fractures of the convexity of the skull, discoverable by the eye, the finger or the radiogram. Difficulties, often insurmountable, are presented when a conclusion in this connection must rest on the focal disturbances of brain functions. The appearance of these symptoms is of course entirely dependent upon the degree of irritation or paralysis that the foreign body produces (if any) or whether it is located in a portion of the brain the physiology of which is unknown. The immediate appearance of symptoms indicates injury by a foreign body.

In this connection, the aid given us by the Röntgenogram is of inestimable value, especially with regard to the presence of projectiles.

Despite the divergent opinions which at various times have been obtruded into the literature of the surgery of the brain, the writer feels justified in subscribing to the conclusion of Chatelin and de Martel,[7] that THE IDEAL THERAPY IN THIS CLASS OF CASES CONSISTS IN REMOVAL OF THE FOREIGN BODY. The reasons for this are summarized by Küttner[6] as follows: The presence of the foreign body interferes with repair and may entirely prevent it; the foreign body is a constant source of concern and is frequently responsible for serious functional, general disturbances or local processes which, after years of time, may destroy the host; it may be responsible for *jacksonian epilepsy,* certain *psychoses* and *late traumatic abscess* and *meningitis.* Küttner[6] makes an exception with regard to very small projectiles which cause little trauma and, unless located in a readily accessible area, may be permitted to remain *in situ.* He adds that when the dangers of removal of the foreign body are greater than those obtaining in a policy of expectancy, the latter should be followed.

In cases of wounds of the cerebral cortex complicated by splintered fracture of the vault of the cranium, primary removal of the splinters, together with all foreign material, and thorough cleansing of the wound are indicated. Macerated portions of brain are best removed with gauze wipes, and bleeding arrested by means of iodoform gauze tamponade. The earlier this is done, the better the prognosis.

In *injuries of the brain not accessible through an open wound,* in which the presence of splinters of bone is suspected, the question of operative relief is not so easily answered. This is determined by the surgeon's conception of the degree of the brain injury, based upon the character of bone injury he is confronted with. This includes splintering of the internal table in simple and restricted compound fractures, stab fractures, gunshot wounds with blind fractures, and small wounds of entrance. When injuries of this sort are located over the motor cortical zone, operative attack is indicated, especially when symptoms of brain irritation are present. Exposure of the lesion is accomplished by trephining (p. 1564). ALTHOUGH PROJECTILES ARE NOT BY ANY MEANS FREE FROM BACTERIA, THE OPERATIVE TREATMENT OF GUNSHOT WOUNDS AND THEIR CHEMICAL DISINFECTION IS NOT ADVISABLE. HOWEVER, TREPHINING IN THIS CLASS OF CASES IS INDICATED FOR THE PURPOSE OF REMOVING THE FOREIGN BODY, INCLUDING SPLINTERS OF BONE, and thus permitting unhampered repair of the lesion. From this standpoint, operative measures are justified, but only when removal of the offending agent does not magnify the already existing trauma to the brain. If this rule is followed, the number of operative cases will be small. *Probing of the canal of a gunshot wound is never permissible.* When the projectile is deeply located, its extraction must be desisted from unless the symptoms presented can be definitely ascribed to its presence. In determining the accessibility of projectiles along the lines stated, the Röntgenogram is of great assistance.

As far as infection is concerned, the primary operative treatment of wounds of the brain is justified when the mechanism of the production of the injury presupposes the entrance of foreign substances. The latter are the conveyors of infection and must (if accessible) be removed. The *technic* of the care of these wounds consists in the extraction of bone splinters, careful cleansing of the wound area, removal of clots and macerated portions of brain tissue, incision of the dura — when retention of wound secretion seems likely — excision of contused soft parts, complete hemostasis, arrest of oozing (by pressure), and, finally, light tamponade of the wound with iodoform gauze. ANTISEPTIC LAVAGE AND CLOSURE OF THE WOUND BY SUTURE ARE TO BE AVOIDED.

The *treatment of gunshot wounds of the brain in war* is still the subject of discussion. Although routine operative measures of relief in all cases were practiced by a number of surgeons during the Boer and Russo-Japanese wars, the conclusion arrived at by a careful study of

the observations recorded by a large number of surgeons serving during the world war may be expressed by the following, quoted from Chatelin and de Martel:[7] "We cannot admit the principle that every foreign body in the brain should be removed. . . . In any event, operation, by dividing fresh nerve fibers, adds to the damage already existing and is likely to add to the patient's troubles. We are of the opinion that, in the case of a foreign body in the brain, no surgical interference is required unless this should be called for by some special symptom pointing to infection." In tangential gunshot wounds of the skull, in which the brain is injured, the operative measures are aimed at removal of splinters of bone and at drainage.

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CHAPTER IV

TRAUMATIC MENINGITIS

When infection occurs in connection with injuries of the contents of the cranium, evidences of inflammation dominate the clinical picture. The *etiological factor* in this contingency, unfortunately very frequent, is the invasion of pathogenic organisms, which, according to Macewen,[1] are chiefly streptococci and staphylococci, occasionally the colon group, and at times the proteus vulgaris. The development of a tuberculous meningitis in connection with trauma seems to have been shown (Küttner[2]). As a rule, the bacterial excitants gain access to the meninges through a wound in the skull. Occasionally the infection is hematogenous in origin. However, even in these cases, local trauma would seem to determine the development of meningitis. Injuries at the base of the skull are frequent ports of entrance.

Traumatic meningitis appears in the form of *leptomeningitis* with the formation of a seropurulent, fibropurulent and purely purulent exudate which fills, to a greater or lesser extent, the meshes and lymph spaces of the pia and often obliterates the gyri and sulci of the brain. At times, it invades the brain substance itself, thus presenting the picture of *encephalomeningitis*. When this process occurs in connection with bloody extravasation (traumatic), ecchymotic areas alternate with the yellow color of the inflammatory zone. The pial inflammation is attended with a similar process involving the dura — *traumatic pachymeningitis purulenta externa* and *interna*.

Traumatic meningeal inflammation may be divided into *early and late meningitis*, depending upon whether the infection begins with the trauma or develops during the process of repair. The former may become manifest within a few hours, the latter may not make its baneful appearance until weeks or months after the injury has been inflicted. Late meningitis is especially likely to develop in connection with retained foreign bodies, necrosed portions of bone and confined necrotic soft parts, wound secretions and trombi, especially of the venous sinuses. So it not rarely happens that an insidious necrotic osteitis or a contused area of the cortex, after a variable period of time, suddenly erupts into purulent meningitis.

The *clinical picture* of traumatic meningitis is rarely presented alone. In the early forms of the affliction, the symptoms usually coexist with those provoked by the concomitant causation, such as concussion, compression, and contusion of the brain, and, in the late forms, the picture is very likely to be blurred by the simultaneous development of encephalitis and traumatic brain abscess. However, the *course* of suppurative meningitis is always acute and, while a restricted process may possibly end in recovery, the rule is that death ensues in a few days.

For this reason, the presentation of a clearly defined clinical picture of traumatic meningitis is hardly possible. If, in connection with a head injury, the patient suddenly develops the symptoms of severe infection with violent headache, wild delirium, rapid pulse and great thirst, and presents a positive Kernig, and this is followed by hyperesthesia of the skin, sinking of the abdomen, stiffness of the neck, convulsions, muscular rigidity, and, finally, stupor and coma, the inference is that he is afflicted with purulent meningitis. This sequence of events is soon succeeded by Cheyne-Stokes respiration and death. The *diagnosis* may be verified by examination of the fluid obtained by *lumbar puncture* (p. 1414).

The *treatment* is really that of prophylaxis and this is encompassed in the aseptic care of wounds of the skull. The comparative rarity of the disease in civil life accentuates the blessings of asepsis.

As the administration of urotropin (2-4 gr. pro die) is followed by its appearance in the cerebrospinal fluid in the form of free formaldehyd, it has been employed as a prophylactic measure against infection in cases of injuries of the brain (Crowe[3]). In cases of moderate virulence, it inhibits the bacterial flora (Denk and Leishner[4]). When the process is fully established, a fatal outcome is certain. In the *circumscribed* form of the disease, the part should be widely exposed with the view to establish drainage. For the purpose, large areas of bone have been removed with a successful outcome in several instances (Macewen,[1] Schlesinger[5] and others).

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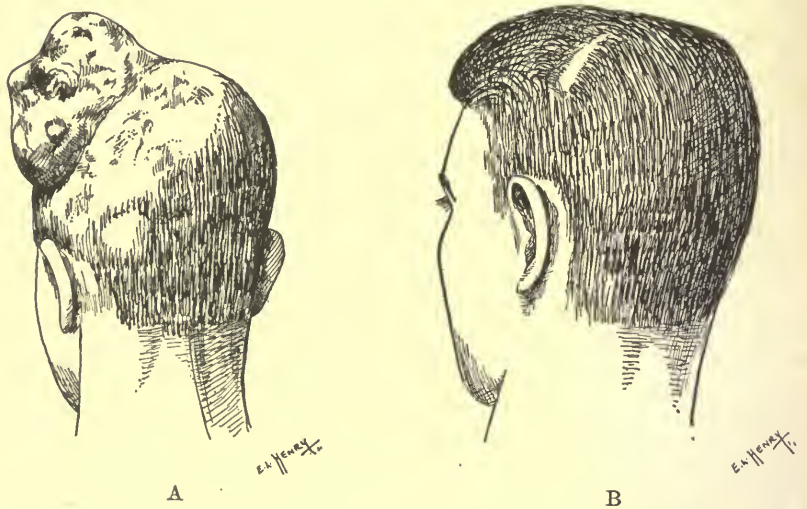
CHAPTER V

HERNIA OF THE BRAIN

The escape of brain tissue from the cranial cavity occurs in connection with wounds involving the coverings of the brain, most frequently in *compound fracture of the vault of the skull,* although, as already stated, brain substance may ooze from a fracture of the base (nose, orbit, and ear). It is most frequently observed in connection with gunshot wounds.



A B

FIG. 748.— *A,* HERNIA OF THE BRAIN IN CONNECTION WITH COMPOUND FRACTURE OF THE SKULL; *B,* SPONTANEOUS REGRESSION AND HEALING.

The *primary extrusion* of brain substance of itself is not always of grave prognostic import, although the location from which it comes has a bearing upon the question of impairment of function. The outlook is very unfavorable when infection occurs under these circumstances.

True hernia cerebri (prolapse) usually develops in the second week following the injury. The pia may cover a part of or the entire pro-

trusion which may reach considerable dimensions (Fig. 748A). The extruded brain soon becomes livid or black from extravasated blood, while its surface softens and becomes gangrenous. If the development of encephalomeningitis is not rapidly fatal, the necrotic portions of brain tissue are cast off and the tumor shrinks. Usually, the shrunken protrusion is ultimately covered with epidermis (Fig. 748B), leaving a pulsating area corresponding to the defect in the skull; this eventually may be covered with an osteoplastic flap (p. 1567).

The *etiology of cerebral hernia* is directly ascribable to increased intracranial pressure. In cases of *traumatism*, this is the result of the accumulation of inflammatory exudate, including intracerebral abscess. In other instances, the protrusion is due to the increased pressure from inoperable *tumors* in which the *decompressive craniectomy* has been performed. This suggests that the latter be not blindly executed.

Küttner[1] calls attention to the appearance (in connection with compound fractures) of the formation of an infectious *hemorrhagic granuloma* which bears some resemblance to hernia cerebri, but is really a fungus-like protrusion of contused brain tissue, provoked by the presence of foreign bodies and splinters of bone. In these cases, ablation of the mass is followed by healing.

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CHAPTER VI

ABSCESS OF THE BRAIN

Localized collections of pus in the brain are divided into:

1. Acute traumatic cortical abscesses.
2. Chronic traumatic deep seated abscesses.
3. Otitic abscesses.
4. Rhinogenous abscesses.
5. Those occurring in connection with carious and ulcerative changes in the ear or other portions of the skull.
6. Metastatic abscesses.

To these may be added the suppurative processes occurring in connection with *acute infectious diseases* (epidemic cerebrospinal meningitis, typhoid fever, scarlatina, measles, influenza, etc.) and those attendant upon the invasion of *ray and other fungi,* tubercle bacilli, and the spirocheta pallida.

Abscesses developing in the course of cerebrospinal meningitis and in actinomycosis are rare (see literature of Oppenheim[1] and v. Bergmann[2]). Circumscribed brain *tuberculosis* and *gumma* present more the picture of brain tumor. The former occurs in connection with caries of the skull of otic origin.

Acute Traumatic Cortical Abscess.—Acute traumatic cortical abscess of the brain occurs most frequently in connection with compound fracture of the skull. In this event, it is likely to be a part of a general leptomeningitis which dominates the clinical picture. When, however, the pyogenic process is limited to a confined cortical area, the symptoms are those of a localized cerebral lesion (muscle spasm, contractures, paralysis, etc.). The development of evidences of inflammation at a considerable period of time (days) after the injury suggests abscess rather than meningitis. The *treatment* consists in enlarging the opening in the skull and evacuation of the pus. Doubt as to the diagnosis does not justify a policy of procrastination, as in leptomeningitis no harm can come from wide exposure of the region of an infected compound fracture of the skull (v. Bergmann,[2] Chatelin and de Martel[3]).

Chronic Traumtic Abscess.— Chronic traumatic abscess of the brain is usually located in the deeper tissues of the medullary substance. It is due to *three causes.* It may develop *sequentially to an acute abscess,* i. e., the greater part of the abscess may discharge externally, the outer tissues heal and the residual infection slowly develop into a chronic abscess. This is especially likely to happen in fractures of the base of the skull. A second cause of chronic abscess is the *retention of an embedded foreign body,* especially a projectile. The third etiological factor in *thrombosis,* which develops in connection with chronic otitis media and osteomyelitis and invades the veins and the skull and sinuses, thus gaining access to the veins of the cerebrum. This occurs in processes that are not due to trauma. To those stated, must be added the rare instances in which an abscess develops at the site of a contusion, the outcome of *contre coup.*

All chronic abscesses of the brain are characterized by a so-called *pyogenic membrane.* Cassirer[4] has shown that this membrane does not act as a demarcation of the process and that the contiguous portions of the brain are often invaded through it. This acounts for the periods of exacerbation of symptoms common to brain abscess. In many instances the restricting deposit of connective tissue is very thin, so that *the abscess either ruptures into the ventricle or upon the surface of the brain* and ends the problem with the picture of diffuse purulent meningitis. An acute abscess terminates in from three to five weeks. After this it takes on the characteristic of the chronic form of the lesion, the persistence of which is very indefinite. Much depends upon its location.

THE DIAGNOSIS OF CHRONIC BRAIN ABSCESS rests on its peculiar course. THE BRAIN SYMPTOMS IMMEDIATELY ASSOCIATED WITH THE INJURY, THE SO-CALLED PRIMARY SYMPTOMS, ARE FOLLOWED BY A PERIOD OF LATENCY WHICH IS MORE OR LESS CLEARLY DEFINED, AND THIS IS FOLLOWED LATER BY SECONDARY AND TERMINAL SYMPTOMS. In traumatic cases this is easily understood. The primary symptoms are the outcome of the lesion produced by the original injury, i. e., concussion, contusion, hemorrhage, etc. When these processes become quiescent, the symptoms that the host develops are dependent upon the location of the abscess. We know now that the conducting elements of the brain may be forced apart by purulent processes of considerable magnitude without destruction of their conductivity, and thus no symptoms are developed. This enlargement occurs intermittently until rupture into the ventricle or on the surface of the brain occurs. The degree

of the primary injury is not in any sense indicative in this connection, being slight in one instance and very severe in another. The *period of latency* is widely variable. At times, this is entirely free from symptoms, at others, attended with irritability, headache, vertigo, mental depression, varying with quarrelsomeness and, not rarely, intermittent somnolence and subconsciousness. In some instances, melancholia and a tendency to suicide develop. *Cerebral edema* is the most common cause of death, next, rupture of the abscess into a ventricle.

The *symptoms following the period of latency* are indefinite and often limited to *neuralgic pain and headache.* Radiation of the latter from the site of the injury and the fact that it is increased in severity by certain movements of the head and body are of diagnostic value. The neuralgic pain is practically limited to the distribution of the fifth nerve. To these are added the symptoms of increasing *general brain pressure,* such as vertigo, vomiting, slowing of the pulse rate, and choked disk. The last is more likely to occur in connection with brain tumor. *Brain abscess may be attended with fever.* However, unfortunately, this symptom of abscess, ordinarily so valuable, is often absent and, as a matter of fact, when present is more indicative of meningitis and encephalitis (Oppenheim[4]). In connection with abscess, fever is not obtrusively present until the process has ruptured on the surface of the brain and has involved its coverings.

Clearly defined focal symptoms are of definite value in the diagnosis of brain abscess, especially when they occur sequentially to headache and fever. Very often the lesion is uncovered by the sudden appearance of *convulsions.* They begin with a cry and a convulsive tremor involving the entire body and are the precursors of a series of brain symptoms. Or, a transitory epileptiform seizure is soon followed by a second and third attack and these are ultimately succeeded by evidence of brain lesion. The focal symptoms are no different from those occurring from other causes.

"When an evening rise of temperature is followed by headache and convulsive attacks, and, later, paralysis of one side of the body develops, the presence of an abscess of the brain is very probable" (Küttner[5]). At this time the behavior of the wound may indicate the presence of abscess, i. e., the granulations are distended and the edges of the wound become edematous, the latter condition frequently extending over the entire scalp. Often fragments of bone are extruded from the opening in the skull. The presence of streaks of pus between the granulomatous masses is indicative of abscess and too often escapes notice.

The *treatment of abscess of the brain aims at the early evacuation of pus,* employing the technic of trephining (p. 1564), if necessary. There is no more imperative indication for the latter than that of affording egress for pus (Küttner[5]). This is justified when there is suspicion of the presence of pus. It is desirable that the end be attained by enlargement of the original solution of continuity of the skull. In all cases the opening must be an ample one. *The return of pulsation in the affected area indicates that the purpose has been accomplished.* During the operation the presence of pus may be announced by discoloration of the dura or by its dull appearance. When opening the dura does not disclose the presence of pus, the brain tissue may be explored with the hollow needle or a thin flat scalpel. Many years ago, Dupuytren[6] employed the latter measure with success. Employment of the Neisser-Pollack[7] brain puncture through the intact skull is permissible, but only if the operator is prepared to open the cranium immediately in the event of the discovery of pus (Oppenheim[4]). The results following the operation are very favorable. Of 77 cases collected by Oppenheim and Cassirer,[4] 56 recovered (see also Lewandowski[8]).

Otitic Brain Abscess.— Otitic brain abscess is of traumatic and otitic origin. Its frequency is made the subject of a comprehensive statistical review by Küttner.[5]

Abscesses originating from the auditory apparatus develop as the result of the extension of chronic inflammatory processes from the ear. Such abscesses occur most frequently in connection with cholesteatomata (p. 1242). These neoplasms destroy the bony structures and provide an avenue of entrance for the infection. As a rule, the process extends directly from the bone, rarely from the mucosa. The chronic suppurative ear diseases concerned in the production of abscess of the brain are (1) those attendant upon cholesteatoma; (2) the putrefactive bone infections; (3) those presenting an intermittent picture with acute exacerbations; (4) those developing from polypoid growths in the tympanic cavity and in the external canal; (5) those originating from perforation of the drum.

It is important to establish (if possible) the presence of the primary suppuration in the petrous bone, as the seat of the abscess corresponds to the location of the suppurative process in the ear. Suppuration in the tympanic cavity which invades the mastoid antrum extends to the mastoid cells, whence it reaches the outer surface, or an extradural abscess in the lateral sinus between the blood vessel and the bone is

formed. This suppuration may produce thrombosis of the vein or it may extend beneath the tentorium and produce an abscess in the *cerebellum.* According to Jansen,[9] cerebellar abscess may also follow suppuration in the labyrinth. Suppuration in the lateral sinus, either with or without sinus thrombosis, leads to abscess in the lateral portion of the cerebellum, while a similar process in the labyrinth leads to abscess in the median lobe. In necrosis of the attic of the tympanum, suppuration may extend along the auditory nerve causing an accumulation of pus above the tegmen (extradural) or it may invade the *cerebrum.* The third temporal convolution and the gyrus fusiformis are most often involved.

The *diagnosis of otitic brain abscess,* like the traumatic, rests mostly on a consideration of the etiological factors. The other symptoms may be arranged in three groups, 1. THE SYMPTOMS INDICATIVE OF A SUPPURATIVE PROCESS; (2) THE SIGNS OF INCREASED INTRACRANIAL PRESSURE; (3) THE EVIDENCES OF FOCAL IRRITATION, OR OF LOSS OF BRAIN FUNCTION. The first consist in the symptoms of fever. However, this is of so low a grade that Macewen[10] considers that a very high temperature argues against otitic abscess. In the second group belong *vomiting* and *headache.* The latter is worse at night and increased by percussion over the site of the abscess (v. Bergmann[2]). Vomiting occurs independently of ingestion and is exacerbated when the patient moves. To these symptoms are added lassitude, inattention, slowness of speech and a tendency toward somnolence. *Slowing of the pulse rate* is characteristic and is more so when headache is at its worst. *Choked disk* is an important sign. In the third group the focal symptoms would be most suggestive, were it not for the fact that they are often absent, and, when present, may be due to epidural suppuration or sinus throbosis. The character of the focal symptoms is dependent upon the seat of the lesion (see cerebral localization, p. 1520).

Otitic cerebellar abscess is not readily diagnosticated. A clearly defined clinical picture is the exception. Much of the cerebellum may be involved before sufficient pressure is made on the worm to produce uncertain gait, vertigo, nystagmus and rigidity of the neck. In the beginning, disease of the labyrinth is usually suspected. By exposing the mastoid process and following the path of infection the location of the abscess may be discovered.

To recapitulate: The question of differentiation arises between otitic TEMPOROSPHENOIDAL OR CEREBELLAR ABSCESS on the one hand, and PURULENT MENINGITIS, INFECTIOUS SINUS THROMBOSIS, AN EXTRADURAL

SUPPURATION, or a SIMPLE EXTENSION OF OTITIS MEDIA SUPPURATIVA TO THE PNEUMATIC ACCESSORY SINUSES, on the other. Brain puncture clears up the diagnosis but, as already stated, this may be employed only when the surgeon is prepared for immediate operative evacuation of the pus in the event of positive findings.

The *treatment* of otitic abscess consists in its evacuation.

Temporosphenoidal abscess is approached in two ways. One is the method of Stacke,[11] in which the abscess is approached through the mastoid antrum. The other, that of. v. Bergmann,[2] is, in all essential regards, similar to the decompressive operation of Cushing (p. 1497). The latter method has the advantage of permitting the extension of the skull section backward to the cerebellum and forward to any desired extent. When the abscess is uncovered, it is incised, and the opening is enlarged with forceps. Tube drainage is universally employed, being maintained until no more pus is discharged.

Rhinogenous Brain Abscesses.— Rhinogenous brain abscesses occur in connection with SUPPURATIVE PROCESSES IN THE UPPER NASAL PASSAGES AND THE ACCESSORY CAVITIES, AND IN THE FRONTAL, SPHENOIDAL AND MAXILLARY SINUSES.

The same endocranial infection occurs in these instances as happens in connection with the ear and its accessory sinuses, i. e., abscess in the frontal lobe, extradural suppuration, meningitis, and thrombosis of the cavernous sinus. The paths by which the infection enters the cranium have been studied by Herzfeld.[12] As a rule, the bony wall of the affected sinus is perforated, but in some instances the infection travels along the olfactory nerve.

It is a well recognized fact that a considerable portion of the frontal lobe of the brain may harbor an abscess without the occurrence of distinctive symptoms and that these do not appear until the process extends to the motor zone (especially the speech center). The sequence of symptoms may suggest the presence of an abscess, especially if the clinical picture of gradually increasing brain pressure is attended with a low grade of fever, and then focal symptoms suddenly occur.

The *treatment of abscess of the frontal lobes* consists in opening the frontal sinus (if necessary) and invasion of the cranial cavity through the posterior wall of the latter. A remarkable series of illustrations showing the widely varying relationship the frontal sinuses bear to the brain is produced in connection with the work of Onodi,[13] who also furnishes a complete bibliography pertaining to the subject.

Abscesses Beneath Ulcerative Processes.— Abscesses beneath *otitic,*

osteomyelitic, carious and ulcerative processes are comparatively rare. On the other hand, any infectious process in the cranial bones may be attended with intracranial suppuration. To this class of cases belong the so-called late traumatic abscesses. These are briefly but sufficiently discussed by Chatelin and de Martel.[3] Cerebral abscesses occurring in connection with tuberculous and luetic bone lesions are also uncommon, yet they do occur (Krause[14]). Chatelin and de Martel[3] saw a number of these cases during the world war, which were sequential to projectile wounds of the brain. The *diagnosis* is based on the focal and general symptoms already taken up, and these, de Martel states, are not as illusive as is generally believed, provided the case is thoroughly studied and complete blood and cerebrospinal fluid examinations are available.

Metastatic Abscesses.— As *metastatic abscess of the brain* has been surgically attacked in the past few years, it presents peculiar interest. It occurs in connection with infectious processes located in more or less remote sectors of the body, especially the intrathoracic organs. Unfortunately, a single metastatic abscess of the brain is rare, several suppurative foci usually being present. Of 22 cases of cerebral abscess collected by Martius,[15] 9 were solitary. The condition is most often associated with putrid and purulent pulmonitis. Claytor,[16] in 58 cases of brain abscess, found bronchiectasis in 29; purulent bronchitis in 10; empyema in 7, which, except one, were the result of gunshot wounds; the rest were due to tuberculosis.

The causative embolus seems to show a predilection for the zone of the Sylvian artery, which Martius[15] explains on a somewhat involved mathematical basis. When the embolus reaches the end of its journey in the motor zone, the clinical picture is that of an acute cortical lesion and this is succeeded by a train of symptoms which do not vary from those already taken up in connection with traumatic abscess.

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CHAPTER VII

THROMBOSIS OF THE INTRACRANIAL SINUSES

Although two kinds of thrombosis are recognized — primary marasmic, and secondary infectious — the latter only possesses surgical interest and will be discussed here.

Infectious Sinus Thrombosis.— Infectious sinus thrombosis is a not unusual and a much dreaded complication of the various purulent processes in the soft parts and the bony structures of the head. It plays an important rôle in the problem presented by the latter, especially with regard to measures of relief.

Sinus thrombosis originates from a number of lesions, such as infection of soft parts and wounds of the bones of the face and skull, phlegmons in these regions, erysipelas, general infectious processes of the mouth, nose and its accessory sinuses, and last, but surely not least, the orbital cavity.

Most feared of all perhaps is the furuncle of the upper lip, the occasional origin of an infection which produces a purulent thrombophlebitis in the angular vein whence it travels by way of the ophthalmic into the cavernous sinus. In not a few instances an apparently innocent dental periostitis of the upper jaw or an inflammatory process in the tonsil is the point of origin of a menacing sinus phlebitis. The process also originates from contiguous brain abscesses and suppurative meningitis. However, the reverse is more often true, and both conditions are the result of the same cause. By far THE MOST FREQUENT CAUSE (in two thirds of the cases) OF SINUS PHLEBITIS IS A SUPPURATIVE PROCESS IN THE EAR (Körner[1]). Although young persons between the ages of ten and twenty years are most frequently attacked (Hessler[2]), no age is exempt. The mastoid portion of the petrous bone is the avenue by which the infection travels from the middle ear to the lateral sinus. The assumption that sinus phlebitis occurs most often as the outcome of *chronic otitis media* has been shown to be an error; the condition is most frequently associated with *acute processes in the middle ear.* The infection is produced by

streptococci, staphylococci, pneumococci and occasionally by the bacillus proteus vulgaris. Besides the sinus infections which extend by the bony tissues, another form, that of *osteophlebitis,* is not uncommon. In this form the infection travels by way of the mastoid venae emissariae. This is especially likely to happen in cases of severe infection of the middle ear associated with so-called *influenza.*

As already stated, the sigmoid portion of the transverse sinus is most often affected. However, primary involvement of the petrous sinus and the bulb of the jugular vein is not excluded. In sinus thrombophlebitis other than the otogenous, that of the *cavernous sinus* is the most frequent. The pathogenesis of the process is taken up in the general part of this work (p. 999).

The *clinical picture* of infectious sinus phlebitis is that of a septic general infection with certain local symptoms.

The *local symptoms* are provoked by the disturbances of the circulation. They are, however, likely to be masked by the inflammation of the surrounding tissues. The cerebrospinal fluid is increased in amount and, as this becomes greater, the symptoms of intracranial pressure, preceded by evidence of cerebral irritation, are more or less rapidly developed. *Headache* is an early and constant symptom; to this is soon added nausea, vomiting and vertigo, i. e., so-called brain symptoms. Differentiation from brain abscess is based on the acuteness of the symptoms, including those indicative of a systemic infection, while clearly focal brain symptoms are lacking. Optic neuritis is usually absent in sinus phlebitis, while in abscess it is often present.

Objective local symptoms, unfortunately not always present, are very helpful. In thrombosis of the transverse sinus, the presence of a circumscribed area of edema at the mastoid process is pathognomonic of thrombophlebitis of the emissaries (Greisinger[3]). This must not be mistaken for the inflammatory edema often found distributed over a suppurating mastoid process. At times deep seated local tenderness may be elicited at the former point and the muscles of the neck are stiff and painful. However, Körner[1] considers that these symptoms are also present in cases of perisinuous suppuration. Voss[4] claims that, when lateral sinus thrombosis is present, the "venous hum" in the jugularis is absent.

When the thrombus extends into the jugularis, the vessel may be palpated as a *hard, painful cord beneath the anterior edge of the sternomastoid muscle,* but a negative finding in this regard is in no sense decisive.

Of much more diagnostic significance are certain evidences of pressure of the thrombosed sinus upon the nerves which pass out of the jugular foramen with the vein, i. e., the vagus, the spinal accessory and the glossopharyngeal. Some of these may be the result of extension of the inflammation to the nerves. The nerve symptoms arising from vagus involvement are hoarseness, dyspnea, and slowing of the pulse. These may cause death. Those of the accessories consist in spasm of the sternomastoid and the trapezius, while impairment of the glossopharyngeal is evinced by inability to swallow and paralysis of the palate. In rare instances, the hypoglossal is involved in the condyloid foramen. Laurens[5] speaks of tenderness over the occiput as of diagnostic value and states that percussion over this zone, even in partially comatose patients, is responded to by what he calls *"cri de la pyémię."*

Körner[1] states that thrombophlebitis of the *petrosal sinus* does not present local symptoms and rarely causes pyemia.

Infectious thrombosis of the *longitudinal sinus* is infrequent. It may develop in connection with otogenous meningitis and by extension from the confluent sinus. It does, however, occur in association with infected compound fractures of the skull, erysipelas or phlegmonous inflammations of the scalp. Infection of this sinus is often attended with dilatation of the veins of the hairy scalp (Lermoyez[6]). According to Gradenigo,[7] an edematous, turgescent area may not infrequently be discovered over the anterior central corners of the parietal bones. He regards this of diagnostic value. Occasionally the eyelids are edematous.

The dominant local symptoms of phlebothrombosis of the *cavernous sinus* consist in a tense edema of the eyelids with chemosis, together with exophthalmus which is caused by retrobulbar edema. Thus is presented a characteristic clinical picture. Pressure on the nerves in this region is evidenced by neuralgic pain in the first branch of the fifth nerve and in paralysis of the muscles supplied by the motor nerves of the eyeball (Hinsberg,[8] Kramm,[9] Marum,[10] Sagebiel,[11] Travernier,[12] Thomson[13]).

While one or more, and perhaps all, of the local symptoms enumerated may be absent, and, in many instances, are only recognized upon careful examination, the general clinical picture of sinus thrombosis is unmistakably that of *general septic infection*. This is extensively taken up in another portion of this work (Part II).

The *diagnosis* of sinus phlebitis rests on the local and the general

symptoms taken into account in connection with the history of a causative infection in the ear, nose, orbit, skull and scalp. After a voluminous presentation, Körner[1] regards the recognition of a hard tender cord in the neck and evidence of pressure on the seventh and ninth nerves as most indicative of thrombosis of the bulb. Of the general symptoms, the clinical picture of a grave general infection is perhaps most determining, especially when this is verified by the blood findings (Leutert[14]). The observations of Oppenheimer[15] are particularly illuminating in this connection.

The *prognosis* of infectious sinus thrombosis may be summed up as fatal in all instances not relieved by operative measures, and of the latter two out of three recover (Körner[1]).

The *operative treatment* of sinus thrombosis owes its inception to Zaufal[16] (1884), its dissemination to Lane,[17] whose daring made the recording of a series of successes possible. The *technic* of the procedure (after primary exposure of the antrum, p. 1655) consists in exposure of the infected sinus. If this is greenish or yellow in color, the diagnosis is clear. The sinus is widely opened and the infected thrombus is removed; bleeding is readily controlled by tamponade. Unnecessary trauma must be avoided. The object of the operation is the establishment of drainage. When the sinus does not present visible evidence of infection, it may be *aspirated*. If fluid blood is not obtained, thrombosis is present. In cases of extensive involvement the sinus may be exposed backward to the confluent sinus. The necessity for LIGATURE OF THE INTERNAL JUGULAR VEIN is not established. The chief objection to the measure lies in the unfortunate fact that it DOES NOT OBVIATE THE ENTRANCE OF SEPTIC EMBOLI INTO THE GENERAL CIRCULATION, which is easy to understand from an anatomical standpoint. In this connection, the review of Haymann[18] is of interest. As far as the operative relief of thrombosis of the cavernous sinus is concerned, this has been attempted by Voss,[4] who employed a technic similar to that used for exposure of the ganglion of Gasser, and by Bircher[19] and Körner,[1] without, however, an encouraging outcome in either instance.

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CHAPTER VIII

EPILEPSY AND ITS SURGICAL TREATMENT

For the purpose of the surgeon it is fitting that the distinction between *genuine and organic epilepsy* be maintained.

It would seem that the experimental work of recent years has established the fact that A LARGE NUMBER OF CASES OF EPILEPSY ARE DUE TO FOCAL LESIONS OF THE CEREBRAL CORTEX. Binswanger[1] has shown that fewer than one half of the cases of epilepsy in childhood are of the genuine variety, and Vogt[2] insists that no case should be condemned to the hopeless class of genuine, or so-called idiopathic, epilepsy until a most thorough examination has been repeatedly made. As observations in this connection multiply, the line of demarcation between the two groups of cases becomes less and less defined. This is especially notable when it is considered that jacksonian epilepsy often merges into the general form and the latter is frequently attended with the characteristics of the former. Both groups are also closely related in an etiological sense. With regard to *congenital epilepsy,* it is not improbable that in a certain number of cases external influences are necessary before the disease is actually developed in a person who is congenitally predisposed in this direction. The parts that congenital malformation and lues play in this are not yet clear.

This varying susceptibility is the reason why we are not in a position to standardize the exciting cause of epilepsy in a quantitative sense. It is impossible to say whether a given injury of the skull or brain will be followed by epilepsy or not. On the other hand, it appears to be certain that aseptic opening of the skull is very rarely followed by epilepsy. Also there is no doubt that the disease is most frequently a sequel of injuries of the motor cortical zone.

It is a disturbing consideration that local lesions are attended with *gradual changes in the entire brain.* While *local cortical lesions* dominate the picture at first, the *general disturbances* soon force these into the background. This occurs most rapidly in the brains of children.

To the surgeon, the most important causes of epilepsy are those changes coincident with *traumatism* and with *inflammatory lesions of the cerebral cortex.* To these must be added *tumors,* which are often heralded by epileptic attacks that not rarely accompany their entire course, presenting the picture of general epilepsy. This is especially true of tumors located in the temporal lobe and involving the contiguous hippocampus major — which is supposed to have some connection with epilepsy. The association of hydrocephalus and epilepsy has been shown by Vogt.[2]

Epilepsy as a sequel to head injuries may be arranged in three categories. To the first belongs the group of cases following injury of peripheral nerves in the coverings of the scalp — usually a scar; to the second, those due to trauma to the bones of the skull or to the dura (for instance, hyperostoses of the intact or fractured skull or adhesions between the cranium and dura); to the third, injuries to the brain itself. The proportion of head injuries that are followed by epilepsy has been studied by a number of writers. Their findings and the experimental work of Westphal, Hitzig and Fritsch are reviewed by Küttner.[3]

While there would seem to be no doubt that head injuries are capable of producing changes in the brain which are followed by epilepsy, it is also true that trauma of other parts of the body may be the exciting cause of the affliction in persons possessing a tendency toward epilepsy. This condition is designated as reflex epilepsy.

In *reflex epilepsy* the causative lesion often involves the trunk of a large nerve, especially the sciatic, or the nerve is attached to the periosteum, or is the seat of a foreign body. Nerves thus affected are exceedingly tender and an attack of epilepsy may be provoked by pressure upon the lesion. In many instances, the so-called *aura* emanates from the area of the involved nerve and it not rarely happens that complete development of an impending attack may be obviated by firmly encircling the central portion of the affected part. On this basis, nerves have been excised and limbs amputated with a not inconsiderable number of successes. Seeligmüller[4] has collected a number of cases of this kind. Reflex epileptic seizures have been relieved by the removal of foreign bodies from the upper air passages, and Frey and Fuchs[5] report cases of epilepsy apparently relieved by the treatment of chronic inflammatory conditions in the pharynx and larynx. The cessation of epileptic attacks following circumcision has been observed by most surgeons. However, the exact establishment of a

causative relationship is not in all instances possible. It is probable that the local cause must be removed before permanent changes have taken place in the brain, i. e., before the epileptic state is fully developed. When the latter is present, attacks are precipitated by minor injuries (Jolly[6]). Given an originally healthy brain, a peripheral injury will not produce epilepsy for many years, but when the disease is once established, an attack is induced by very slight irritation (Unverricht[7]). According to v. Bergmann,[8] scars located in the scalp are more likely to be followed by epilepsy than are those situated elsewhere.

Compound fractures of the skull, especially the depressed variety, are frequently followed by epilepsy, although this also occurs as a sequel to fracture with loss of substance. In both instances, the convulsions are ascribable to *changes in the tissues of the brain.*

The theory of so-called *cortical epilepsy* is based on the fact that irritation of the cortex of the brain is attended with spasm on the opposite side of the body. This was demonstrated by Hughlings Jackson[9] (1861) and its bearing on the study of epilepsy was brought out by Charcot,[10] who coined the term *jacksonian epilepsy.*

A large number of *operative measures* directed toward the relief of jacksonian epilepsy have been employed with varying success. These cannot of course be reviewed here. In standardizing the value of a procedure it must be remembered that during a certain period of time following *any operation, epileptics* are likely to be free from attacks. At least three years must elapse, free from attacks, before a case may be regarded as permanently relieved. In other cases, improvement may be claimed, if the attacks are less frequent and less severe.

The aim of the present day operative treatment is the removal of injured bone, together with the damaged cortex beneath it. The possibilities in this connection were indicated by the case of v. Bergmann[8] (1887), who extirpated the hand center in a case of traumatic epilepsy. Since then the method has become *en vogue,* principally as the outcome of the labors of Krause.[11]

However, even these efforts are attended with many failures. Recently the results have been improved by a more exact *identification of the brain area giving rise to the primary spasm.* This is accomplished by means of a mild faradic current. After preliminary double ligature of the vessels leading to the identified part, the cortex is excised to a depth of 5 mm. (Fig. 749). The immediate effect of this procedure is more or less transient paralysis of the part supplied by the excised

cortex. This is ultimately overcome, provided there is no infection of the wound (Friedrich[12]). The causative relationship between comparatively slight head injuries and epilepsy, especially in children, has been shown by Friedrich,[12] whose observations have been verified and data amplified by Weil.[13]

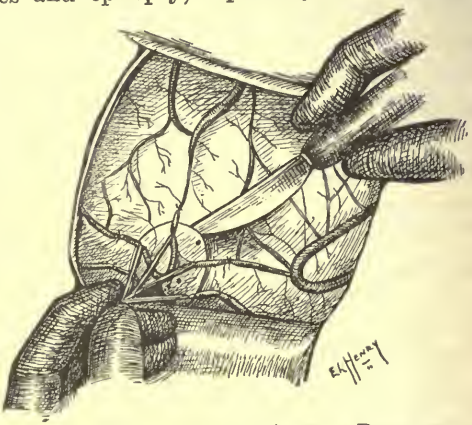

FIG. 749.—EXCISION OF AREA OF PRIMARY SPASM FOR RELIEF OF JACKSONIAN EPILEPSY.

In traumatic jacksonian epilepsy the operation must be performed early. However, it is to be remembered that THE DISEASE IS NOT DUE TO THE INJURY BUT TO ITS CICATRIZATION and that trauma added to the primary lesion is, therefore, objectionable. In cases of epilepsy due to a defect in the skull, closure by means of an osteoplastic flap is often followed by relief.

Relief in cases of jacksonian epilepsy by operative measures is possible. They are of no avail, however, in genuine epilepsy. The assumption of Kocher,[14] that increased intracranial pressure is responsible for the changes the brain undergoes in epilepsy, is supported by the fact that the manometer shows the former condition to be present during an attack. On this basis, exposure of the brain is made, in all instances, by means of a large osteoplastic flap (Fig. 752). At this writing, most surgeons (after the cortical extirpation) excise the dura and close the defect by a free fascia and fat transplant (from the patient's thigh), upon which the osteoplastic flap rests. Recent clinical observations, including those of the writer, justify continuance of the method.

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CHAPTER IX

MENTAL DISEASES FOLLOWING CRANIAL INJURIES AND THE SURGICAL TREATMENT OF MENTAL DISEASES

A statistical review of the causal relationship between head injuries and mental diseases compiled from various observers shows that insanity is ascribable to head injury in about 3 per cent of the cases. Perhaps the work of English[1] is the most illuminating in this connection, available at this time. Injury to the head of the infant during birth is probably responsible for the largest number of cases. It is true that severe deformity of the fetal head is in most instances completely recovered from. When, however, the birth injury is responsible for a MENTAL DISTURBANCE, the latter ALWAYS APPEARS IN THE FORM OF IDIOCY AND IS MANIFEST DURING THE FIRST YEAR OF LIFE. Of course not all cases of congenital idiocy are due to head injuries. In arriving at a conclusion, the surgeon must take into account congenital chronic hydrocephalus, early meningitis, congenital cerebral defects, and microcephalus (Wulff,[2] Vogt[3]). In the *adult,* the form of *mental derangement* following injury is *inconstant* and presents a number of characteristics. However, all of the psychoses present certain elements in common, i. e., *disturbances of memory and lack of mental concentration.*

The *causative trauma* may be a severe one, i. e., extensive destruction of the skull and its contents or a gunshot wound of the brain; in other cases it may be so slight in degree that external evidences of injury are absent. A normal brain is capable of withstanding a severe injury, while one weakened by alcohol, etc., shows the evil effects of comparatively slight trauma. In all instances, the effect of trauma is expressed by unconsciousness, which results from concussion (*commotion psychosis*). The anatomical lesion of these psychoses may consist in gross changes, such as hemorrhages, areas of softening, scars, traumatic cysts, or in more *minute changes,* such as occur in the *blood vessels of the cortex* in the form of transient and permanent dilatations of blood vessels, scleroses, punctate hemorrhages, infiltration of the

sheaths of the vessels, etc. The influence of all grades of trauma in the production of psychoses has been recently studied by Roussy and L'Hermite[4] in connection with the world war. This presentation is worthy of perusal. These indefatigable workers recognize the influence of predisposing factors, such as heredity, and lay especial stress on lues and alcohol. They also furnish a comprehensive bibliography pertaining to the subject.

Lunacy after head injury may be divided into two groups. In the first, the psychosis is immediately sequential to the injury, and in the second, it does not occur until a greater or lesser period of time has elapsed.

In cases of the *first group* the injury is attended with severe concussion and unconsciousness, which lasts for a variable period of time (often for weeks). When consciousness returns, the patient has no recollection of the injury and memory in general is especially distorted. A so-called *traumatic retrograde amnesia* is common, so that the patient does not recall events that happened before the injury; the past is, as it were, "wiped out." Westphal[5] reports cases of definite forms of psychosis attended with *aphasia, agnosia, and apraxia.* In others, the patient is irritable, maniacal, has hallucinations, and is unable to sleep, a condition that Wille[6] calls *delirium traumaticum.* As a rule, return to the normal occurs in three or four weeks, although permanent impairment of memory is not rare.

A number of cases of *acute traumatic psychosis* present a clinical picture closely resembling the so-called Korsakoff *symptom complex* (Heilbronner,[7] Kalberlah[8]), in which the patient is unable to fix his attention, memory is impaired, and appreciation of time and place is disturbed. In addition to this, the patient is easily excited, remembers things that did not happen (false memory), and attempts to supply something when he cannot remember. In this type the prognosis is not good and is worse when the primary stage of unconsciousness is immediately followed by a so-called *posttraumatic dementia* (Köppen[9]).

In the *second group* of cases are those so-called *soul disturbances* which develop some time after infliction of the head injury. However, the actual appearance of the psychosis is often preceded by a *prodromal period* which is characterized by disturbances of sensation, changes in the patient's temperament and character, and lessening of mental capacity. Of these, the CHANGES IN CHARACTER AND HABITS, IN CONTRAST TO THE PREVIOUS CONDITION, ARE MOST MARKED (TRAUMATIC PSYCHOPATHIC CONSTITUTION). Peace loving persons become quarrelsome,

moderate drinkers imbibe excessively, or are intoxicated by small quantities of alcohol. Many of these cases go on to secondary idiocy, others develop melancholia, catatonia or mania. The causative relationship between the injury and the second group of cases is certain, if the prodromal stage has been presented and this is attended with clearly defined brain symptoms (Küttner[10]).

The class of cases in which a head injury is slight and simply renders the individual more susceptible to the development of a psychosis cannot be discussed here. It is, however, proper to state that a relationship of this sort is of clinical significance only when a distinct connection between the injury and disturbances of the sensorium can be shown.

Injuries of the head have been held responsible for *general progressive paralysis.* Modern serological examinations seem to have shown that injury of the skull of the non-luetic is not followed by general paresis; on the other hand, it is not improbable that *injury of the skull in a luetic is followed by an exacerbation of the paretic condition.*

It occasionally happens that a patient who has apparently completely recovered from a head injury and is again "engaged in the contest of life" will gradually develop certain disturbances grouped under the head of *"traumatic neurosis."* The genuine traumatic neurosis is characterized by so-called brain symptoms, i. e., headache, vertigo, mental depression, lack of ambition, lack of concentration and impairment of memory. The patient is likely to complain of palpitation of the heart and dizziness, especially when he "bends over." In some of these cases lumbar puncture has revealed increased cerebral pressure which would indicate an underlying pathological causation. In others, the symptoms are purely functional (vasomotor). According to Küttner,[10] traumatic neuroses of the latter sort occur most frequently in individuals deeply concerned in the award of a disability pension and when the award is made the condition usually disappears.

The *operative treatment of traumatic psychosis* has been frequently attempted. Unquestionably favorable results have been obtained in cases of psychic epilepsy (Heidenhain[11]). This is also true of cases of progressive idiocy following epilepsy in children (Krause[12]). The technic consists in the osteoplastic repair of cranial defects (p. 1567), the removal of cicatrices, cysts and tumors of the brain. Linear cranial resection enjoyed only a brief period of popularity (Köppe,[13] Schüller,[14] Borchardt,[15] Semelaigne[16]).

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CHAPTER X

THE SURGICAL TREATMENT OF BRAIN TUMORS

As the outcome of the collaboration of the neurologist and the surgeon the localization of a brain tumor is verified by operative exposure in 75 per cent of the cases (Bruns[1]). Of these, it is possible radically to remove only a certain number.

Tumors of the brain are divided as follows:

1. *Genuine tumors.*
   (a) primary.
   (b) secondary.
2. *Granulation tumors.*
   (a) tubercle.
   (b) gumma.
   (c) actinomycosis.
3. *Diseases of blood vessels* attended with the clinical picture of tumor.
4. *Cysts.*
5. *Parasitic cysts.*

Of the *genuine tumors, glioma* and *sarcoma* are the most common (Allen Starr[2]). Their histology is taken up in Part VI (see also Oppenheim,[3] Starr,[2] Henschen,[4] Küttner,[5] Auvray[6]). *Psammoma* is regarded as a form of sarcoma (p. 1290).

Enchondromata, lipomata, dermoids, and *teratomata* are rare tumors of the brain. *Angiomata* are taken up on page 1158.

Of the *granulation tumors, conglomerate or solitary tubercle* is by far the most common. Starr[2] says "they form 50 per cent of all tumors of the brain in children." *Gumma* is more common than is histologically proved. Oppenheim[3] explains this on the ground of their frequent spontaneous disappearance. *Actinomycosis* is very rare (see page 404).

Diseases of blood vessels that provoke the clinical picture of tumor belong to the class of telangiectases, cavernous and racemose angiomata (Krause[7]).

Cysts of the brain are frequently the outcome of degenerated genuine tumors; true cysts also form in this situation (for instance, a dilated fourth ventricle may provoke the symptoms of a tumor of the acusticus). Cysts of the pia develop occasionally after trauma, or are inflammatory or syphilitic in origin (*meningitis chronica circumscripta serosa*). The accumulation of fluid in the posterior cistern may be mistaken for a cyst formation.

Parasitic cysts appear in the form of the *cysticercus cellulose* and *echinococci*. They are usually multiple and may be located in any portion of the brain.

The *etiology* of tumors of the brain is as little understood as is that of new growths in general (p. 1094). The *influence of trauma is of practical importance.* In *tubercle* and *gumma,* trauma is not the direct cause, but may determine localization of the process, on the *locus minor theory.* That trauma is responsible for the formation of a genuine tumor is highly improbable; the injury may call attention to its presence. On the other hand, injury may be responsible for the cyst formation and for basal aneurism.

Of the *symptoms of brain tumor* it may be said, that while a tumor may be present in the brain and life complete its cycle without the development of symptoms ascribable to the new growth, as a rule the presence of the lesion is evidenced by more or less typical disturbances of brain function.

The *general symptoms* are in no sense in accord with the size of the tumor. They appear especially early in growths involving the posterior cranial fossa; on the other hand, those located in the central convolutions and pons do not provoke symptoms until they attain considerable dimensions.

The general symptoms may be divided according to their clinical importance as follows: (1) *headache;* (2) *choked disk* (disturbances of vision); (3) *psychoses;* (4) *convulsions;* (5) *vertigo;* (6) *vomiting;* (7) *changes in pulse and respiration.*

Headache is probably never absent, although the presence of brain tumor has been established before this symptom developed (Horsley[8]). While the headache is constant, *violent* exacerbations are frequent and these are often attended with befogged consciousness. An exacerbation, i. e., congestion of the brain, may be precipitated by alcohol, mental excitement, coughing, defecation (Bruns[1]). The *character* of the headache is described as dull and boring, and may be localized or general. A violent attack of headache may be provoked by *percus-*

sion of the skull. In some instances percussion elicits a *tympanitic note* (*bruit de pot félé of Bruns*[1]), which is regarded as indicative of thinning of the cranial bones, and often corresponds to the site of the new growth.

Choked disk is of especial value in the diagnosis of tumor of the brain in all stages of the lesion and is rarely absent. Vision itself need not be impaired, but gradually this is also affected and is attended with scintillations alternating with periods of darkness. Blindness occurs late but may appear suddenly, and unfortunately usually affects both eyes. In tumors of the cerebellum, choked disk appears very early, while changes in the optic nerve may be absent.

In the early stages of tumor development *psychic disturbances* are not marked, but later on they become more clearly defined. Of these, mental hebetude is the most marked. The patient does not evince interest in his surroundings and responds to inquiries only when they are persisted in. Somnolence is a frequent symptom and the facial expression is usually stupid. In a certain number of cases genuine psychoses are developed (melancholia, delirium, and mania). However, Bruns[1] and others hold that in these instances the tumor simply precipitates a latent psychosis. Psychoses are not dependent upon the seat nor upon the character of the tumor, but are most likely to develop in connection with growths attended with hydrocephalus interna. However, not a few authors regard the development of psychoses as indicating a lesion of the frontal lobes (see Oppenheim[3]).

Convulsions occur in 25 to 30 per cent of cases of brain tumor (Bruns[1]). They are likely to resemble genuine epilepsy, so that the affected persons may be regarded as epileptics for years before the diagnosis is made. The seizure closely resembles jacksonian epilepsy in character.

Vertigo is an inconstant and not very helpful symptom in tumor of the brain and is rarely present in its genuine rotary form, except in lesions involving the vestibule, the acusticus, pons, medulla, and the fourth ventricle. In these instances it is usually associated with nystagmus. The feeling of dizziness is increased by changes of posture and, when the head is suddenly passively moved, the patient is likely to fall (Bruns[1]). According to Hitzig,[9] tumors of the frontal lobes may be attended with vertigo.

Vomiting may be regarded as an expression of general cerebral pressure. In tumors of the cerebellum emesis is ascribable to direct irritation of the "vomiting center." Despite the vomiting, the appe-

tite continues good. Its concurrence with headache causes confusion with nephritis. Küttner[5] relates an instance in which he was asked to decompress a case of this sort, but fortunately the diagnosis of nephritis was established in time.

Slowing of the pulse is a vagrant symptom of brain tumor. Oppenheim[3] says that rapid pulse is as frequent as a slow one, except, of course, when general cerebral compression is established. Distinctive *respiratory disturbances* develop only in tumors of the posterior cranial fossa and then consist in the arhythmia and the *Cheyne-Stokes* type. Additional general symptoms are those of general cerebral pressure.

The *focal symptoms* of brain tumor relate to the disturbances of function consequent to destruction of sectors of brain and the damage done to the tissues contiguous to them. The symptoms thus produced are either the result of an *immediate* influence on brain tissue or of *remote or distal* disturbances. While the former are of distinct diagnostic value, the latter often lead the surgeon astray. The effect of the destruction of definite regions of the brain is taken up on p. 1520.

Although, as a rule, a case of brain tumor comes to the surgeon after the diagnostic problem has been studied by the neurologist, the ophthalmogist, and the otologist, it is necessary that the findings thus obtained be carefully weighed; and their application to the surgical problem from the surgeon's standpoint must decide the feasibility and the justification of making operative efforts at relief. In recent years the *Röntgenogram* and the employment of *brain puncture* have been found of great assistance in recognizing the presence and location of brain tumors. The radiogram has been of signal service in connection with tumors of the hypophysis, and Henschen[4] reports that in cerebellopontian tumors, widening of the bony canals of the internal ear has been recognized in the same way.

The *Neisser-Pollack*[10] *brain puncture* gives positive evidence, not only of the site, but also of the character, of the tumor. The procedure is not without certain elements of danger, but these are not unsurmountable. The value and the dangers of *lumbar puncture* are already taken up (p. 1414). Neither Cushing[11] nor Borchardt[12] punctures the spine in these cases, unless the skull is opened, a precaution to which the writer has already alluded and herewith reiterates as imperative. Distention of the cerebral ventricles with air and subsequent radiography (*ventriculography*), recently introduced by Dandy,[13] seems to possess considerable value.

In the *treatment* of tumors of the brain, *medicinal therapy*, even of

the palliative form, is of restricted value, with perhaps the exception of gumma. In this connection, the surgeon is warned that diagnostic antiluetic treatment must not permit time — valuable to operative methods of relief — to be wasted, especially as temporary improvement of symptoms has followed antisyphilitic medication in cases of genuine brain tumor (Oppenheim,[3] Redlich[14]). As far as salvarsan is concerned, the writer can only reiterate the warning sounded by Ehrlich,[15] that its employment in cases of brain tumor is likely to do great harm. Headache is relieved only by opiates.

The *general technic of the extirpation of brain tumors* is similar to that of trephining (p. 1563). The question of the one or two stage operation may be answered in favor of the latter, except when the tumor is extradural or in cases in which the lesion is diagnosticated very early and cerebral pressure is not marked. In this connection the statistical data furnished by Krause[7] are instructive.

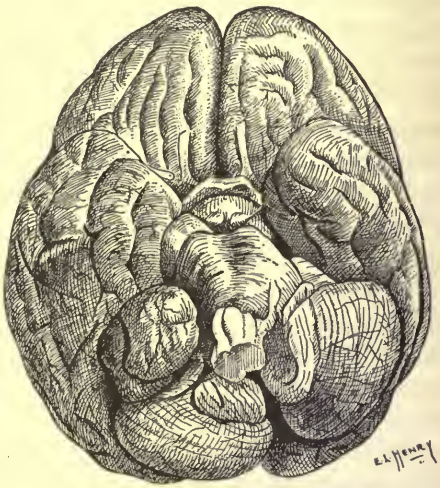

Fig. 750.— Tumor of the Cerebellopontine Angle.

The special considerations that must be impressed upon the student in connection with the technic of operative attack upon tumors of the brain relate to the posture of the patient, the selection of the anesthetic, and the method of opening the skull. The dura is usually opened in the form of a flap. When the tumor is located, it is removed with a dull spoon.

Subcortical tumors present especial difficulties, although their presence may be revealed by elevation of the cortex, by flattening of the convolutions, and by obliteration of the sulci. Palpation is not of great aid, needling may be; and while removal of a cylinder of tissue for immediate microscopical examination is determining, incision of course is certain.

Extirpation of the tumor is followed by more or less *bleeding;* it is usually considerable. Divided vessels are ligated, but this must be done with as little trauma to the brain tissue as possible. Transient

tamponade of the cavity (iodoform gauze) is often necessary; the gauze must be inserted with just enough pressure to arrest the bleeding. The dura should be sutured into place when possible.

Exposure of the posterior cranial fossa, because of the relative frequency of tumors in this region deserves special mention. At the present time the operation is frequently performed under local anesthesia. Krause[7] and Borchardt[12] employ the osteoplastic method (Fig. 752); Küttner, and not a few others, sacrifice the bone. The former technic is desirable but involves greater danger of injury to the medulla than does the latter.

Decompressive trephining as a palliative measure of relief in inoperable tumors of the brain is an established procedure. The operation consists in opening the skull and the dura. Most surgeons sacrifice the bone; Krause[7] fashions an osteoplastic flap, outlining it by means of wide liberating bone sections. In either event, the edges of the bone should be covered with dural flaps in order to prevent laceration of the brain, which immediately protrudes through the opening of the skull. When the tumor is located, the opening is made over it. In other instances the skull is entered over the left temporal lobe in left handed people, and over the right in right handed people. Cushing[11] advocates *subtemporal decompression.* In lesions of the cerebellum the posterior cranial fossa may be decompressed in the manner employed for approaching this area for other purposes.

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CHAPTER XI

THE SURGERY OF THE HYPOPHYSIS CEREBRI

The first operative attack upon the hypophysis in the human was made by Horsley[1] in 1906; the subsequent surgery of this mystifying organ is based on the work of Schloffer.[2]

A detailed discussion of the functions of the hypophysis is of course impossible here. The conclusions of Cushing[3] may be regarded as authoritative in this connection. They may be briefly stated as follows: The hypophysis is essential to life; its total extirpation is followed by death; so is excision of its anterior lobe alone; removal of the posterior lobe alone is not followed by serious results. *Partial removal* of its anterior lobe leads to peculiar disturbances, i. e., adiposis, sexual regression, polyuria and glycosuria; these are similar to the conditions attendant upon tumors of the gland and are designated in the literature by the term *dystrophia adiposogenitalis. Pituitrin* is derived from the posterior lobe. Extracts from the anterior lobe do not seem to possess the value of those of the posterior. The "physiological correlation" of the pituitary body and other internal secretion glands (thyroid, testicle, ovary, etc.) is still a subject of investigation. Its clinical significance is already shimmering on the horizon (see bibliography of Melchior[4] and the experimental work of Crowe, Cushing and Homans[5]).

Of the lesions of the hypophysis, only the tumors are of importance to the surgeon. These are divided into two general groups, the *adenomata,* and the so-called *tumors of the hypophyseal anlage of Erdheim.*[6] The latter represent an especial form of epithelial growth, partly carcinomatous in character, and originate from displaced embryonic elements. *Adenomata* of the hypophysis are benign tumors responsible for the clinical picture generally known as *acromegalia* (p. 1073). On the other hand, the embryonic tumors of Erdheim[6] are regarded as standing in a causative relationship to *dystrophia adiposogenitalis* (Fröhlich[7]).

The *local symptoms* of tumors of the hypophysis relate primarily to

disturbances of vision due to pressure on the *optic chiasm,* of which narrowing of the temporal field of vision is regarded as typical (*bitemporal hemi-anopsia*). This is usually associated with progressive amblyopia, the outcome of optic nerve atrophy, which ultimately goes on to complete amaurosis. *Choked disk* is not an essential symptom, nor is paralysis of the motor nerves of the eyeball common (see Uhtoff,[8] Kanavel[9]). *Headache* is a constant symptom (Frankl-Hochwart[10]). In some instances, persistent *hydrorrhea* developes (Kütt-

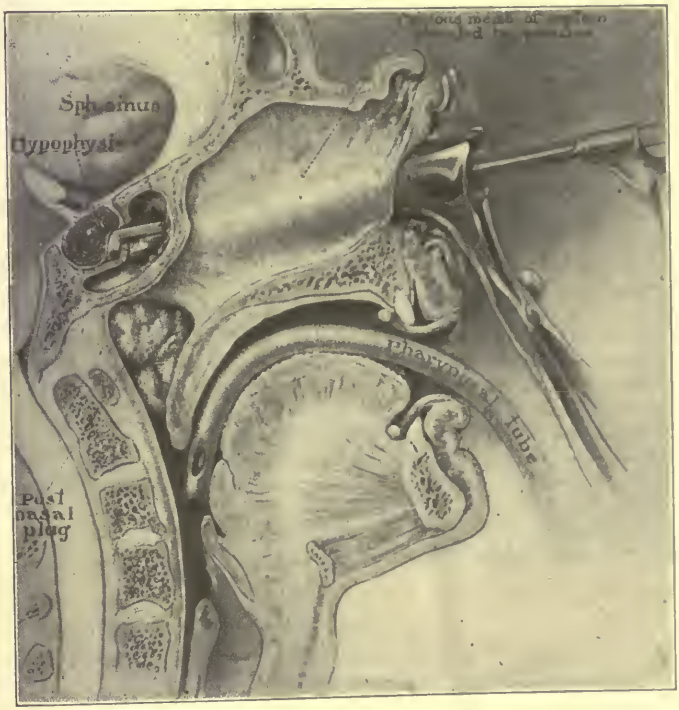

FIG. 751.—HYPOPHYSECTOMY (Kanavel).

ner[11]). The *Röntgenogram,* which reveals enlargement of the sella turcica, is of great value.

The *general trophic disturbances* of hypophyseal tumors belong in part to the domain of acromegalia and in part to dystrophia adiposogenitalis. In acromegalia, trophic disturbances may be absent (Uhtoff[8]). According to Benda,[12] *acromegalia* is the outcome of *hyperfunction* of the gland, while *dystrophia adiposogenitalis* is the result of *hypopituitarism.*

The indication for *operative effort* at relief is based on the *local symptoms,* especially impairment of vision.

Schloffer[2] turned the nose aside; v. Eiselsberg[13] elevated the nose and approached the gland through the frontal sinuses; Cushing[3] also formed a frontal osteoplastic flap. Recently, Kanavel[9] raised the nose, resected the septum and gained access to the sphenoid from below (*infranasal route*). Figure 751 gives an adequate notion of the last method. The endonasal method of Hirsch[14] is ingenious and no doubt is readily accomplished by an expert rhinologist. Of fourteen cases, only two died. The improvement following the operation in cases of acromegalia is likely to be little short of startling.

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CHAPTER XII

THE TECHNIC OF TREPHINING, BRAIN PUNCTURE, RESECTION OF THE SKULL, CRANIECTOMY, CRANIOPLASTY AND DURAPLASTY

In recent years the efforts of a number of surgeons have been devoted to devising methods of invading the cranial skull, with the result that "the classic operation of trephining with its instrumentarium, inherited from the dark ages of several centuries ago, has quite properly been relegated to history" (Küttner[1]). As a rule, the cranium is opened for the purpose of applying measures of relief to its contents and the opening in the bone is made as close to the site of the lesion as is possible. Occasionally, removal of a sector of the skull is essential for the purpose, but ordinarily this is unnecessary unless the bone itself is injured or diseased. The latter class of cases includes primary and secondary resection of splinters of bone and excision of bone tumors, tuberculosis, lues, etc.

Therefore, for the purpose of applying intracranial methods of relief, the approach is accomplished by the so-called *temporary* or *osteoplastic resection of the skull* originally devised by Wagner[2] and carried to its greatest extent by Doyen[3] (*hemicraniectomy*).

The technic of opening the skull may be divided into the following groups:

1. *Simple drilling of the skull.*
2. *Simple resection of the skull.*
   a. *The classic trephining with the trephine.*
   b. *Chisel resection.*
3. *Osteoplastic resection of the skull.*
   a. *The method of Wagner.*
   b. *The method of Doyen and allied methods.*
   c. *The method of Toison and Obalinski.*

Simple Drilling of the Skull.— Simple drilling of the skull is done for the following reasons: (a) to withdraw fluid from the ventricles; (b) for intraventricular medication; (c) for diagnostic purposes. The last has been developed by Neisser and Pollack.[4] At this time intra-

cranial medication is restricted to the injection of *antitetanus serum.*

The technic of the procedure is simple. Most surgeons use an electrically driven drill. In order not to have to grope for the drill hole, Götze[5] bores the skull with a "gutter drill' and uses it as a guide for a thin wire; the hollow tube is introduced over the wire. The puncture is occasionally followed by a pial hemorrhage. However, this is rare.

Simple Resection of the Skull.— In simple resection of the skull, the *classic trephine* is used only when a definite object is to be attained and when this may be accomplished through a small opening in the skull (2.5 to 3 cm.). Krönlein[6] ardently supports the use of the trephine for ligature of the middle meningeal artery and in cases of sharply defined lesions of the bone, such as punctured fractures. The means by which the trephine is operated is immaterial.

Resection with the chisel is employed in the same conditions as is the trephine. At the hands of the skilled technician it possesses a distinct field of usefulness. However, the concussion coincident with the use of the mallet is objectionable.

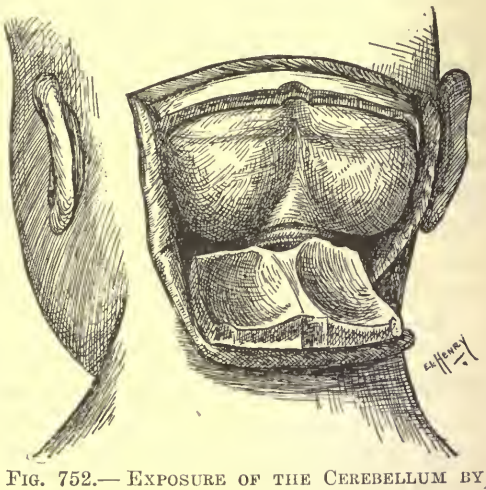

FIG. 752.— EXPOSURE OF THE CEREBELLUM BY THE OSTEOPLASTIC FLAP METHOD.

Osteoplastic Resection of the Skull.— Osteoplastic resection of the skull is the method of choice for the purpose of *draining an abscess, extirpation of a tumor, removal of a foreign body, and exposure of an epileptogenous focus.*

The essential steps in the technic consist in incising the scalp and periosteum in the outline of a flap; this is followed by section of the bone in the line of the incision in the soft parts. The osteoplastic flap thus outlined is then fractured at its base and the parts turned down (Fig. 752). Various methods of dividing the bone have been devised. The primary opening of the skull which permits the introduction of bone cutting instruments may be accomplished by means of the Doyen[3] "fraise sphérique" already described (p. 787). Of the hand driven

bone cutting tools in use today, the Dahlgren[7] craniotome (Fig. 753). is the most satisfactory. Perhaps the "last cry" in this connection is the electrically driven "automatic trephine" of de Martel[8] (Fig. 754).

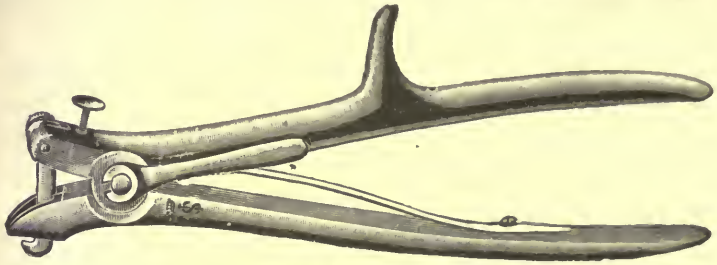

FIG. 753.— THE ORIGINAL DAHLGREN CRANIOTOME.

This ingenious instrument makes the necessary number of primary openings in the skull and is so arranged that injury to the underlying dura is impossible. The bone flap is then cut with the spiral osteotome (a modification of Sudeck's[9] device) (Fig. 755), which is used in connection with the same apparatus. The motor is described on page 784. The *rotary circular saw* of Salzer,[10] extensively used by Doyen,[3] is an effective instrument but is difficult to control. When the three sides of the bone flap are cut, its base is fractured by means of a periosteal elevator or other suitable bone lever. It is permissible to add that special bone cutting instruments that meet the particular notions of the surgeon are readily constructed when the principle of the procedure is borne in mind and an electric motor is available. For instance, Borchardt[11] uses a sort of "bone plow" to cut the external table and completes the section with the Sudeck[9] spiral osteotome;

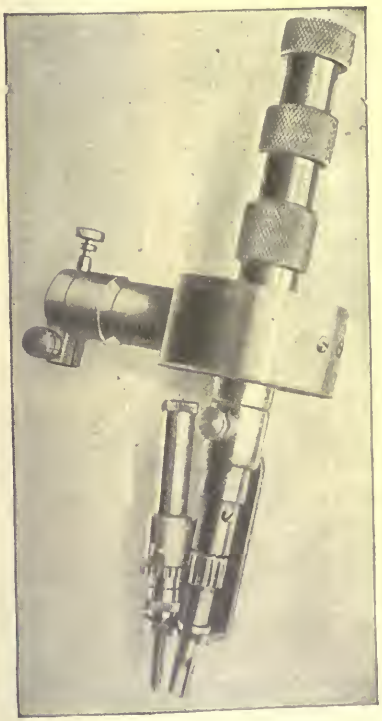

FIG. 754.— DE MARTEL'S AUTOMATIC TREPHINE.

Krause[12] uses the Dahlgren[7] craniotome entirely and regards electrically driven instruments as a "fad." It is certainly unwise to be entirely dependent upon them. Most surgeons use a spherical burr for the purpose of making the primary openings; yet this is readily accomplished with a small trephine.

Toison[13] of Lille (1891) is responsible for the use of a flexible saw which divides the skull from within. This was soon displaced by Obalinski,[14] who showed that the Gigli[15] wire saw was better adapted to the purpose. Although the danger of injury to the dura and the brain involved in the use of the wire saw was largely overcome by the interposition of various materials (Braatz,[16] Lauenstein,[17] and Gigli[15]), the method fell into disuse. However, recently de Martel[8] reports having used it in a number of cases. Most American surgeons use the spherical burr of Doyen[3] for making the primary openings in the skull and cut the bone with the Dahlgren[7] (called Devilbis in this country) craniotome; or the de Martel[8] trephine and spiral osteotome driven by the electric motor previously described are used throughout. The latter is used in all well equipped hospitals.

FIG. 755.—SPIRAL OSTEOTOME.

Bleeding from the scalp may be controlled in a number of ways. These include the use of the Kredel[18] plates, the Heidenhain[19] sutures, and the specially constructed clamps of Makkas.[20] The last are most satisfactory and have dislodged the first two from most clinics.

Bleeding from the emissaries and the veins of the diploe may be arrested by the introduction of wooden or ivory plugs, or Horsley's wax may be used (p. 42). Crushing the open vessel in its bony bed with a rongeur or a sharp blow with a mallet often accomplishes the purpose. Krause[12] uses an especially constructed bone hook; Sauerbruch[21] applies negative pressure to the body, thus lessening the quantity of blood in the brain.

The Closure of Cranial Defects.— Notwithstanding a few recorded instances (of doubtful accuracy), defects in the skull do not undergo spontaneous ossification. In addition to this, the connective tissue repair is not of sufficient strength to afford, in any sense, the protection to the cranial contents necessary to its welfare (Küttner[1]). Not the least objectionable feature of the presence of a defect is the trauma that the unprotected brain substance is constantly subjected to as a

part of the ordinary activities of life (Koenig[22]). It is, therefore, incumbent upon the surgeon to prevent the occurrence of defects and to repair them when present. The first effort of this sort is recorded by Koenig[22] (1890), who closed a large defect in the skull (traumatic) by means of a pedunculated skin-periosteal-lamina-externa flap derived from the contiguous skull. Müller[23] had advised the use of the method in lieu of the osteoplastic craniectomy, and for this reason the term *"Müller-Koenig osteoplastic flap method"* is used in the literature. The value of the procedure is unquestioned, not alone in traumatic cases, but also in congenital defects and in the repair of loss of substance sequential to disease (tumors, tuberculosis, lues, etc.). The flap is fashioned in accord with the general principles of plastic

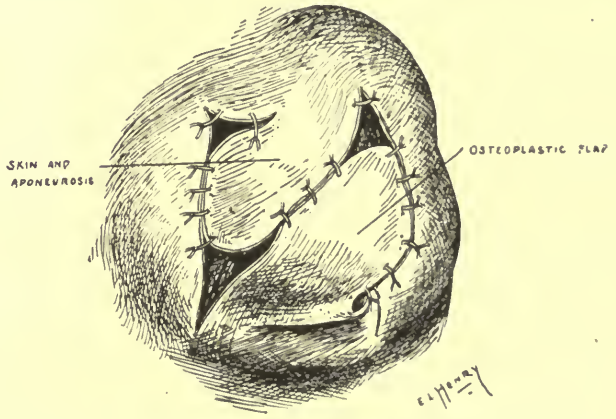

FIG. 756.— OSTEOPLASTIC REPAIR OF DEFECT IN SKULL.

surgery and is best separated by means of a broad, thin chisel; or a fine, thin, flexible saw may be used.

Cranial defects have been closed by many other means with varying success. Of these the following are employed more or less frequently:

Reimplantation of the excised trephine disk; reimplantation of small fragments of bone; implantation of bone derived from various sources, such as the tibia of the patient, that of another person, or that of one of the lower animals; implantation of decalcified bone; implantation of boiled and raw untreated dead bone; implantation of metal and celluloid plates and the implantation of filigree net (see Küttner[1]).

Duraplasty.— Plastic repair of dural defects is made with a view to

obviating adherence of the brain to the soft parts. The following table of the various materials employed for the purpose is taken from Küttner[1]:

A. *Alloplastic* — 1. rubber, gutta percha; 2. celluloid; 3. gold; 4. silver; 5. platinum.

B. *Heteroplastic* — 6. egg membrane; 7. fish bladder; 8. wall of a blood vessel.

C. *Homoplastic* — 9. peritoneum.

D. *Autoplastic* — 10. periosteum; 11. fascia; 12. fat; 13. skin.

The relative value of these agents is not definitely established. It would seem, however, that, as is the case in all plastic surgery, autoplastic methods hold out the best hope of success, and of these the implantation of a free fascia and fat transplant is perhaps the most satisfactory (v. Eiselsberg[24]). The recent contribution of Brüning,[25] who separates a layer of the contiguous dura by injecting it with salt solution and folding the outer layer over the defect, is worthy of consideration. It is said to be easily carried out for the purpose of closing small defects.

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CHAPTER XIII

CRANIOCEREBRAL TOPOGRAPHY

The relationship the regions of the brain bear to the surface of the skull varies with age, size, sex, and race of the individual, and is, therefore, not susceptible to exact mathematical laws. To the surgeon this means that in invading the brain, he must expose sufficient of its surface to allow for these variations. When, in accord with certain topographical rules, the site of bone section is determined and the brain is exposed, further information as to the identity of certain areas is obtainable by means of electrical stimulation of the cortical areas (see Krause[1]).

Topographical location is based on the determination of the situation of the *central fissure* and the *fissure of Sylvius* with respect to the surface of the skull. A number of methods have been devised for the purpose. Of these, the one of Krönlein,[2] known as the *Krönlein construction,* based on the studies of Froriep,[3] is the simplest and most satisfactory.

The lines upon which the construction is based are as follows (Fig. 757):

1. *The base line* — linea horizontalis auriculo-orbitalis.
2. *The upper horizontal* — linea horizontalis supraorbitalis.
3. *The anterior vertical* — linea verticalis zygomatica.
4. *The middle vertical* — linea verticalis articularis.
5. *The posterior vertical* — linea verticalis retromastoidea.
6. *The linea Rolandi* (oblique).
7. The *linea Sylvii* (oblique).

The points of orientation for the projection of 1 to 5 are as follows:

1. The base line through the inferior rim of the orbit and the upper edge of the meatus auditorius.
2. The upper horizontal through the superior edge of the orbit, paralleling the base line.
3. The anterior vertical from the middle of the zygoma, perpendicular to the base line.

4. The middle vertical for the head of the inferior maxilla, perpendicular to the base line.

5. The posterior vertical from the posterior edge of the base of the mastoid process, perpendicular to the base line.

The two oblique lines are *secondary* and are drawn as follows:

6. The *linea Rolandi* connects the point of junction of the anterior

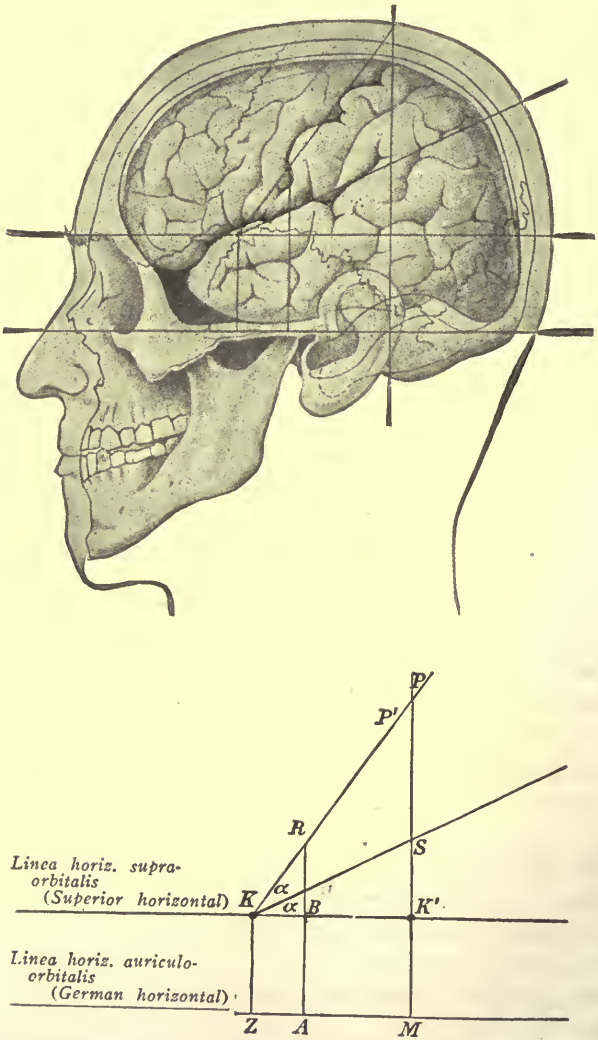

FIG. 757.— DIAGRAM OF LINES AND ANGLES CONSTRUCTED UPON THE SKULL FOR THE LOCATION OF THE CONVOLUTIONS AND FISSURES (Krönlein).

vertical and the upper horizontal lines with the point where the posterior vertical crosses the vertex of the skull.

7. The *linea Sylvii* bisects the angle formed between the linea Rolandi and the upper horizontal line and prolongs the line of bisection posteriorly until *it crosses* the posterior vertical line.

In the construction (Fig. 757) this is indicated by:

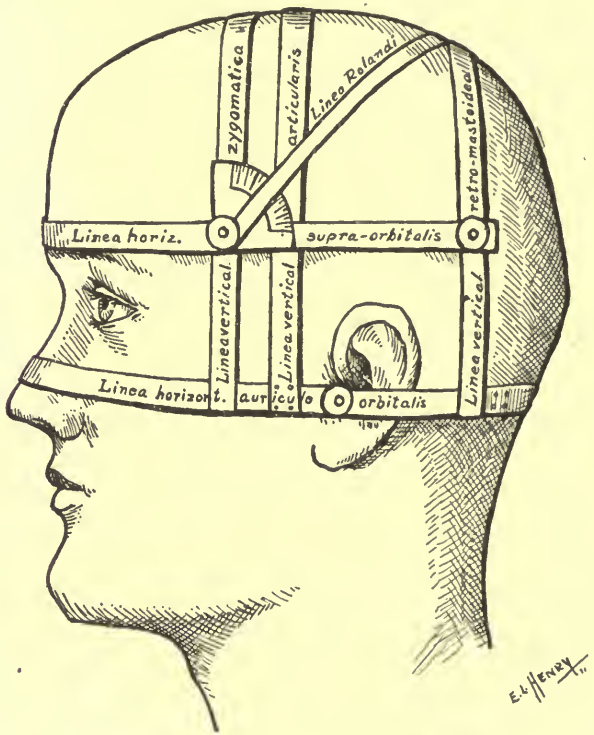

FIG. 758.— KRÖNLEIN'S CRANIOMETER.

k. The point of bifurcation of the Sylvian fissure.
s. Upper end of fissure of Sylvius.
r. Lower end of the fissure of Rolando.
p. Upper end of the fissure of Rolando.
k.k. Krönlein's two sites for trephining.
a.b.k.m. v. Bergmann's resection points.

Outlining of the construction on the scalp is facilitated by the use of the Krönlein[4] craniometer shown in figure 758.

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CHAPTER XIV

THE SURGERY OF THE EAR

Congenital Malformations of the External Ear.— Congenital malformations of the external ear (excluding deviation from the artist's idea) are rare. Joseph[1] classifies those susceptible of plastic correction as follows:

I. *Deformations of the ear.*
  A. *Abnormal size of the ear* (macrotia).
    1. Of the cartilage.
    2. Of the lobule.
      a. Abnormal width of the lobule.
      b. Abnormal length of the lobule.
  B. *Abnormal form of the ear of normal size.*
    a. Flattening of the rim.
    b. Triangular ear.
    c. Fissures of the lobule.

II. *Malpositions of the ear.*
  A. Abnormal protusions of the ear.
    1. With soft cartilage.
    2. With hard cartilage.
  B. Rolling in of the ear.

III. *Adhesions of the ear.*
  A. Synechia of the rim with the tragus.
  B. Synechia of the lobule with the cheek.

Otomiosis is practiced for the purpose of reducing the size of the ear. Figure 759 shows a method which would seem sufficiently elastic to meet most contingencies. *Reduction of the size of the lobule* is easily accomplished by excision of a wedge shaped portion and suture. The *triangular ear* may be corrected in a similar manner. In *fissure of the lobule* the edges of the split are refreshed and apposed by suture.

Abnormal protrusion of the ear (otapostasis), *when the cartilage is*

soft, is easily overcome by removal of an elliptical sector of skin from the junction of the auricle with the scalp and suture of the wound. *When the cartilage of the ear is hard,* a sector of this tissue must also be removed (Keen,[2] Koenig.[3] See also bibliography of Joseph No. 1).

Otosynechia is readily corrected by following the principles of plastic surgery already discussed (see Goldstein[4]).

Paraffin therapy has been attended with so many accidents and is so frequently followed by unfavorable results that it has a very narrow field of usefulness.

Injuries of the Ear.— The exposed position of the auricle renders it particularly liable to injury. On the other hand, its vascularity and

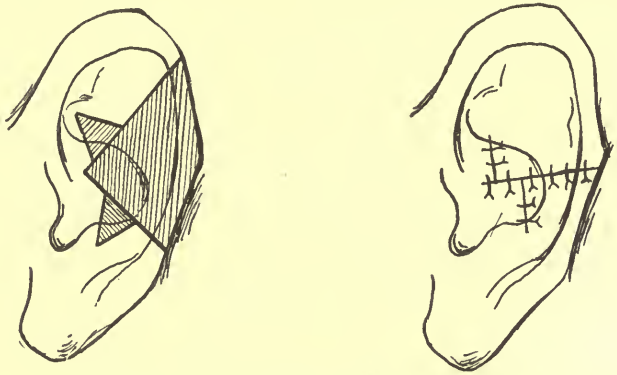


FIG. 759.— REDUCTION OF OTOMEGALIA.

the resistance on the part of the cartilage, explains the fact that wounds in this situation heal rapidly and are rarely infected.

Contusions of the auricle are often followed by the formation of *hematomata,* which are peculiarly persistent. Repeated trauma causes grave deformation of the auricle (prize fighters, wrestlers, etc.). *Primary incision* and evacuation of the extravasated blood has a tendency to prevent the deformity.

Injuries of the external meatus are important because of the subsequent cicatrical stenosis. In this event, the secretions collect behind the obstruction and give rise to serious disturbances; the retention of a small quantity of material interferes seriously with hearing. In most instances some form of plastic operation is necessary for relief.

Fractures of the articular surface of the maxillary joint may give rise to considerable bleeding and cause the formation of a hematoma

involving the external auditory canal. The introduction of hot fluids (acids, hot steam, etc.) may destroy the canal and drum and even gain access to the middle ear. In these instances, the area is so painful that removal of the foreign substance must be accomplished under narcosis.

Foreign bodies in the external auditory canal are often introduced by children (button, pea, bean, etc). However, occasionally, a portion of an instrument used for the purpose of removing wax is broken off and left behind. It is important that efforts at extraction of the foreign body do not force it still farther into the canal. Once the body is forced beyond the constriction where the cartilaginous and bony portions meet, extraction is difficult. In these instances the foreign body rests in the recess at the anterior medial part of the canal, and, if it is small (such as an insect), may be entirely hidden from view.

When the *foreign body is located in the cartilaginous* canal, it may often be easily forced out by means of a gentle stream of warm water propelled by a syringe; or the canal may be illuminated with a head mirror and a small dull hook used for the purpose. Narcosis is desirable. An astonishing amount of injury has been produced (even by physicians) by forcible efforts to remove foreign bodies from the ear, especially when exploration is made on the suspicion of the presence of the body. In these cases, the drum has been punctured and the bones of the middle ear "fished out" by mistake. The surgeon is admonished that a slight delay is not a disadvantage and that no effort should be made to remove the unwelcome visitor until proper conditions obtain, i. e., a good light, narcosis, and the proper instruments. When the *foreign body is deeply located* and cannot be removed by the means stated, it is best to make a curved incision behind the auricle, displace it forward and deliver the offending agent. It may be necessary to remove the posterior edge of the bony meatus. However, this is preferable to traumatizing the drum or worse.

Injuries of the drum are the result of *direct* or *indirect* violence. Of the *indirect injuries,* those produced by sudden variations in air pressure, such as occur in connection with an explosion, a blow, a kiss, etc., are the most frequent. At times the drum is not entirely ruptured but is simply the site of a minute hematoma. The lesion has occurred as the result of sneezing or coughing, and occasionally follows forcible inflation of the middle ear (Politzer bag). A special form of rupture of the ear drum occurs in connection with diving into

water, the water gaining access to the middle ear and provoking a more or less severe inflammation. In professional divers, enclosed in an air tight apparatus, the drum is ruptured as the result of the air pressure, especially when the eustachian tube is stenosed so that equalization of the pressure in the middle ear does not occur promptly. Rupture of the drum in connection with fractures of the base of the skull has already been taken up (p. 486).

Injuries of the drum by *direct violence* are usually produced by the accidental or intentional introduction into the auditory canal of various instruments, such as a toothpick, a wisp of straw, etc. Occasionally, an instrument is forced inward to the wall of the labyrinth and gains access through the foramen ovale to the labyrinth itself, or it may enter the cranial cavity through the tegmen tympani. Injury of the labyrinth is immediately heralded by vertigo, nausea, and subjective disturbances of hearing. As a rule, the patient falls to the ground. After a few days or weeks, the more stormy symptoms subside and, usually, ultimately disappear. Invasion of the labyrinth through an already infected middle ear is followed by serious inflammation of the former, which is likely to be fatal. When recovery ensues, the hearing, on the injured side, is permanently lost.

The *diagnosis* of ruptured tympanum is not always easy, at first, the collection of blood in the canal interfering with the examination. REMOVAL OF THE BLOOD BY MEANS OF THE SYRINGE IS NOT PERMISSIBLE. Excessive quantities of blood may be gently removed with cotton wipes. When the bleeding has ceased and the danger of infection is over, the extent of the injury may be readily determined with the aid of the speculum and the head mirror.

The *treatment* of traumatic rupture of the ear drum is essentially that of *noninterference;* even extensive lesions heal spontaneously. Meddling only interferes with repair. The normal mucosa of the middle ear is sterile, and, therefore, manipulations tending to force bacteria through the rupture into the former should be avoided (Kümmel[5]). The ear should be lightly tamponed and the loose gauze plug frequently changed. It is perhaps wise to cover the ear with an aseptic protective dressing. When infectious meningitis develops, which is likely to happen when an already infected middle ear is traumatized, the cells of the antrum must be opened and free drainage provided for.

Gunshot wounds of the ear are attended with grave disturbances of the auditory apparatus, especially when the projectile is fired di-

rectly into the meatus. The labyrinth may be destroyed or merely placed out of function by the concussion coincident with the injury. In the latter event, recovery from the disturbances ultimately ensues. In many cases the temporal bone is badly comminuted and the base of the skull presents fissures more or less remotely located from the site of the bullet; the *facial nerve* is often destroyed or injured.

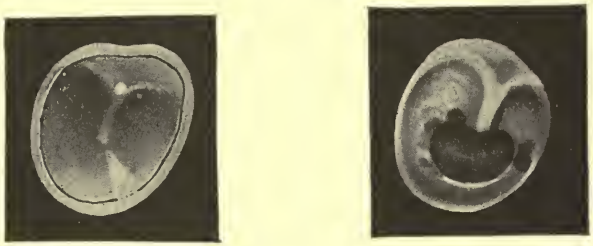
As retention of the projectile is always, sooner or later, followed by suppuration, it should be removed. Determination of its exact location by means of the Röntgenogram is of great assistance. The technic of extraction may involve extensive surgical manipulations and the attempt should be preceded by a careful study of the problem.

Inflammation of the Ear Drum.— Inflammation of the ear drum usually occurs as a part of otitis externa or media. It is, however, subject to isolated inflammation in the course of acute infectious diseases, especially in association with so-called influenza (Kerrison[6] doubts this). This form of infection is called *"myringitis bullosa"* and is characterized by the formation of small vesicles located upon various sectors of the membrane. The vesicles look like mother-of-pearl and contain a clear serum, at times mixed with blood. The condition must be *diagnosticated* from middle ear inflammation. The *treatment* consists in careful opening of the vesicle or vesicles.

Acute Otitis Media.— Acute otitis media develops as a part of a general systemic infection, or is due to the extension of an inflammatory process from the pharynx through the eustachian tube. Hematogenous infection of the middle ear (Moos[7]) occurs in association with all acute and chronic infectious diseases. The variation in the underlying causative factor probably accounts for the widely divergent character of the otic complication. There would seem to be no doubt that mild forms of otitis media without the formation of purulent exudate frequently occur. Whether these are bacterial in origin, or are simply due to obstruction of the eustachian tube, establishment of a vacuum in the middle ear, and the development of *hydrops ex vacuo*, is a disputed question (see Kerrison,[6] Moos,[7] Politzer.[8] In any event, if the inflammation does not soon subside, the process taken on the form of a purulent otitis media, which may go on to serious consequences. In these cases the drum, which, at first, is injected, and is violet in color, becomes red or copper colored; previously existing depression (caused by the vacuum in the middle

ear) is abolished and, as the exudate in the cavity increases, the membrane bulges perceptibly. The protrusion of the drum may be circumscribed, being limited to its posterior upper quadrant, or may involve the anterior upper part (Shrapnell's membrane) alone. In the *circumscribed form,* restitution to the normal is often effected. However, perforation is not rare and may occur at the lower or at the upper aspect of the membrane. In the former event, evacuation of the exudate is followed by more rapid recovery than is rupture of the upper sector. In the *diffuse form* the perforation may occur at any sector of the membrane and in severe cases multiple perforations are not uncommon.

Of the *symptoms, pain* is the most dominant, even in mild cases; it usually begins suddenly and is likely to be very severe. It is described as boring, throbbing and stabbing in character, and radiates



A B

FIG. 760.—*A,* NORMAL TYMPANUM. *B,* PERFORATED TYMPANUM (Reik).

to the temporal and mastoid regions. Tenderness over the entire region, especially over the mastoid, soon develops. This is characteristic in children, in whom the disease is very common. The patient complains of "roaring, ringing, and pulsation in the ears;" occasionally loud noises cause pain and the spoken voice is reduplicated (autophonia). Audition is soon reduced.

The *general condition,* in mild cases, is unaffected. As the process progresses, the clinical picture soon presents the characteristics of *an acute infectious process.* When the drum ruptures, the symptoms subside with astonishing rapidity, provided the opening establishes sufficient drainage. If, however, the perforation happens to be at the upper aspect of the drum, the symptoms are likely to recur.

The *course* of the disease varies widely. If suppuration does not occur, the process subsides in a few days; if pus forms, the inflammation may persist for weeks or longer. Not a few acute processes merge

into the chronic form of the disease. Kümmel[5] says that the latter contingency may often be avoided by appropriate treatment during the primary acute disease.

The *complications of acute otitis media* are as follows:

1. Severe inflammation of the accessory pneumatic spaces — the recessus epitympanicus, and especially the antrum and the cells.
2. Destruction of the bony walls of the tympanic cavity or exposure of portions of, or of the entire, auditory ossicles with subsequent periostitis or necrosis.
3. Extension of the inflammation to the labyrinth.
4. Involvement of the contiguous venous blood vessels in the temporal bone.
5. Involvement of the brain and its membranes.

Inflammation of the Accessory Pneumatic Spaces.— In every case of intense otitis media the lining of the recessus epitympanicus, the mastoid antrum and the cells is involved. In most instances the process heals simultaneously with that in the mesotympanum. Not infrequently, however, grave changes take place in the accessory pneumatic spaces.

The presence of large cells and certain constitutional pecularities, such as diabetes, predispose to infection of the mastoid spaces. According to Körner,[9] the infection does not always originate in the middle ear, but is often due to an "osteomyelitis petrosa," and the mastoid cells are secondarily involved.

Insufficient drainage through the middle ear (through the tube) is the most frequent cause of inflammation of the accessory cavities. If, during an inflammation in the middle ear, the pneumatic sinuses are invaded and the former is suddenly drained, the process subsides in both situations. When, however, the infection is intense in character, the means of communication are not sufficiently patent to establish an exit for the secretions. This results in one or more of the following:

1. The connections with the tympanic cavity ulcerate, the mucous membrane trabeculae are destroyed, and gradually sufficient drainage is established so that spontaneous healing occurs.
2. Though the process in the pneumatic spaces persists, the bacterial excitants diminish in virulence and a chronic discharge of purulent exudate into the middle ear is established.
3. The vascular communications between the antrum and the periosteum of the mastoid process convey the infection from the former to the latter, where a periostitis develops. The process in the

mastoid cells goes on to the formation of minute abscesses; the bony walls of the canals undergo more or less destruction, so that additional communications with the antrum (fistulae) are established. Occasionally, a considerable portion of the mastoid process undergoes sequestration.

When periosteal suppuration is established, it may perforate externally (mastoid fistula). In this event, the symptoms of inflammation subside and spontaneous healing may follow. However, this is the outcome only when a large cortical sequestrum is expelled and the spontaneous opening is a large one. In most instances, secondary proliferation of granulation tissue, in an unsuccessful effort to eliminate the sequestrum, destroys more or less extensive areas of the temporal bone, often as far as the tegmen tympani, or extends backward, involving the posterior cranial fossa.

Perforation of the suppurative process medially into the digastric fossa is not so rare as external rupture. This is accomplished through a large cell located at the medial side of the mastoid process (Bezold[10]). Rupture of the abscess is likely to be followed by a deep cellulitis of the neck, which at first may simulate phlebitis of the jugular vein. However, as a rule, a suppurative cervical adenitis follows.

The upper wall of the auditory apparatus may also be involved and become necrotic, and sectors of the bone may be spontaneously expelled. As a rule, however, it is necessary to approach this region by surgical means and to remove the necrosed bone with gouge, chisel, etc.

4. The most baneful outcome of the infection occurs when retained pus and granulations gain access to the dura or labyrinth. The process either perforates the tegmen tympani and gains access to the middle fossa of the skull, or enters the sigmoid fossa by way of the posterior mastoid cells. In acute otitis media the latter is most commonly the case. Involvement of the labyrinth occurs frequently in connection with chronic inflammatory processes.

5. A certain number of cases of virulent middle ear infection are attended with general septic infection. According to Körner,[9] the infection gains access to the circulation through a number of small veins in the bones (*osteophlebitic pyemia*). Leutert[11] believes that the infection enters through the sigmoid sinus.

Kümmel[5] divides mastoiditis as follows:

1. *Diffuse, suppurative inflammation of the mucous membrane lining the hollow spaces of the temporal bone,* in which the antrum

and the larger cells are filled with purulent exudate. The process is usually a mild one, but is very likely to become chronic.

2. *Inflammatory infiltration of the lining of the hollow spaces of the temporal bone,* in which the cells of the mucous membrane are swollen and the bone is softened and necrosed. This often occurs in connection with very acute infections. There is a little pus in the antrum and not infrequently an extradural abscess is present. In this form the clinical symptoms are very severe.

3. *Osteitis of the temporal bone* occurs occasionally with but little involvement of the hollow spaces. In this form the primary lesion is probably in the bone.

In most instances mastoiditis is secondary to otitis media and the former takes on the character of the latter. Form 1 may be so mild as to cause the surgeon to hesitate in regard to operative measures. However, the tendency to chronicity, even in this class of cases, should be taken into account, especially when it is remembered that an acute exacerbation is likely to develop at any time.

The *prognosis of mastoiditis* is to a considerable extent influenced by the promptness with which operative efforts at relief are made. Early operation prevents the development of serious complications in most instances. Spontaneous healing is very rare. Most cases, except in tuberculosis and diabetes, heal in from four to six weeks following the operation.

1. The *clinical manifestations of mastoiditis* are moderate in form 1, and not so severe in the others as those attendant upon the provocative otitis media. Pain is described as boring, tearing, deeply located, and is often referred to the temple. Tenderness is marked, especially over the planum. Fever is variable and is indicative of the character of the infection. The general symptoms are those of an infectious process.

The *diagnosis of mastoiditis* is not always easy, at first. When otitis media is attended with tenderness, its increase is significant and swelling is diagnostic.

An erroneous diagnosis may be arrived at in connection with phlegmonous otitis externa, cellulitis of the parotid gland or of the neck. In doubtful cases incision is justified. A coëxisting otitis media is of course helpful in every regard. Persistence of fever after the drum has ruptured is a suspicious circumstance.

2. *Ulceration and necrosis of the bony walls of the tympanic cavity*

and ossicles are a part of the clinical picture of suppurative otitis media.

3. *Labyrinthian disease* occurs when infection travels through the fenestrae. Habermann[12] has shown that the bones of the internal ear may be involved in an osteomyelitic process. The affliction is characterized by a loss of the sense of balance, dizziness, vomiting and nystagmus. In these cases the doom of the function of hearing is sealed. In some instances the infection extends along the fibers of the acusticus or through the aqueductus cochleae into the arachnoid cavity or through the aqueductus vestibuli into the sacculus endolymphaticus; in both instances meningitis or a serious phlebitis develops.

4. *Infections of the sinus and the bulb of the jugular vein* occur as the result of extension of middle ear inflammation by way of the small vessels (phlebitis) leading from the mucosa of the mastoid cells or as the result of an extradural abscess which extends through the wall of the sinus. The other cranial sinuses may be involved by extension of the thrombus from the transverse sinus. The bulb may be directly invaded through the floor of the tympanic cavity, and the caveronous sinus may be infected by the same route.

Of the infections of the cranial contents by extension from the middle ear, extradural abscess, especially in the sigmoid fossa, is most common, although suppuration above the tegmen tympani also occurs. The process gains access to the cranium either as the result of destruction of the bone, or by way of the lymphatic and blood vessels. In a certain number of cases the infection travels in the connective tissue surrounding the vessels leading from the antrum. Suppurative processes of this sort are likely to be responsible for leptomeningitis. Brain abscess in connection with otitis media is rare.

The *treatment of otitis media* in mild cases should be directed to correction of the nasal and pharyngeal conditions which interfere with drainage of the middle ear. In the more severe cases it is customary to apply so-called *antiphlogistic measures.* The ice bag often increases pain; the use of a "cooling" rubber tube coil is more satisfactory. The application of a leech over the mastoid process is of doubtful utility. *Early paracentesis* is the treatment of choice. The patient is narcotized, the drum is exposed by means of an ear speculum, and is widely incised at its anterior inferior aspect. *Local anesthesia* of the drum may be achieved in from twenty to thirty minutes by a soft cotton pack soaked in concentrated cocain solution or a solution of equal parts of phenol, menthol and cocain. The pain of otitis media

may be controlled by the application of a 5 to 10 per cent carboglycerin solution. This is kept in contact with the drum by a light cotton tampon. Too frequent or prolonged use of the agent causes sloughing of the soft parts. After a paracentesis or spontaneous perforation of the drum, the canal should be cleansed by frequent, gentle lavage with boroglycerin, etc. Bier's hyperemia (p. 249) is of doubtful value. Vaccine therapy is still an indefinite quantity. Delay in opening the mastoid cells is not permissible. If simple incision does not afford relief, the antrum must be opened.

Chronic otitis media, when primarily of low grade, is secondary to processes of similar character in the pharnyx and in the region of the opening of the eustachian tube. It possesses importance in its influence upon hearing and has no surgical significance. Probably many of the cases are luetic and tuberculous in character. MANY TIMES THE CONDITION IS A SEQUEL OF INFECTION OF THE PHARNYX AND MASTOID CELLS WHICH HAVE NOT BEEN TREATED. In some instances the process shows an intermittent character (*chronic recurrent otitis media*) and is very likely to "flare up" into an acute intracranial or menacing labyrinthian complication. However, most cases of chronic otitis media go on to sclerosis with the formation of adhesions. In this class of cases the discharge of pus through the perforated drum varies widely. The secretion often emits an offensive odor.

The *prognosis* in chronic middle ear disease, from the surgeon's standpoint, involves the danger — only too frequent — of abscess of the temporal loeb, sinus phlebitis, cerebellar abscess, etc.

The *treatment* is of course directed toward the relief of the underlying cause. Mechanical cleansing of the tympanum is imperative. This often calls for the exercise of considerable manipulative skill, especially when it is necessary to remove granulomatous tissue from portions of the tympanic cavity difficult of access. It is important that the latter be kept dry; Politzer[8] uses alcohol for the purpose. The therapy of this class of cases has become a very highly specialized art (see Kerrison,[6] Moos,[7] Politzer[8]).

Tuberculosis of the middle ear occurs in connection with all stages of pulmonary tuberculosis and is often complicated by a mixed infection. It is characterized by rapid destruction of the auditory apparatus in the absence of the clinical picture of inflammation; the facial nerve is often paralyzed early in the disease. In children under seven years of age, a considerable percentage of cases of mastoiditis is tuberculous in character. Henrica, quoted by Körner,[9] regards most

of these cases as primary bone infection — a view which Görcke[13] contests. See also Schwabach[14] and Nos. 6, 7, 8 of the bibliography.

Syphilitic pharyngitis often causes obstinate middle ear disease (Habermann[15]). *Actinomycosis* of the middle ear has occurred (Zaufal[16]).

New Growths of the Ear.— Granulomata occur in connection with all chronic (occasionally with acute) forms of otitis media. Their development is induced by persistent irritation of the mucosa, such as that produced by foreign bodies, secretion from the middle ear, hairs, cotton shreds, epithelial scales, cholesterin and lime crystals, necrotic bone, etc. The small tumors are covered with a layer of epithelium, the larger with a coating of pavement or cylindrical epithelial cells. When they protrude into the auditory canal, they become polypous in form and often reach gigantic proportions and protrude from the meatus. Although they are likely to recur, they should be removed. This is best accomplished with the snare, after which the stump is cauterized with chromic acid or the galvanocautery.

Genuine fibromata of the external ear are rare. *Keloids* occur in connection with perforations of the lobule for earrings. These often attain great size, especially in the negro race. They should be extirpated. Recurrences after complete removal and *aseptic healing* are not common. So-called fibromata of the auditory canal are probably granulomata into which connective tissue had been deposited.

Angiomata involve the external ear, especially in children, as a part of growths on the cheek. They are regarded as congenital in origin (fissural angiomata) and appear in the telangiectatic, plexiform, and cavernous forms (see Angiomata, p. 1158).

Chondromata present no regional pecularities, nor do the various kinds of *osteomata.* Almost all of the other benign and malignant tumors found in other portions of the body appear in the region of the ear and are subject to the same laws as regards diagnosis and treatment (see Tumors, p. 1141).

Pearl tumors, which resemble dermoid cysts, possess especial interest. A certain number of observers attribute their origin to the presence of cheesy pus, around which layers of epithelial cells are deposited; others consider them to be genuine tumors (see Panse,[17] Schwartze[18]). The former theory is the more widely accepted. Their *clinical significance* lies in the resemblance they bear to a low grade of inflammatory process.

Another form of growth, *resembling cholesteatomata,* consists in a

collection of masses of epithelial cells in the antrum or in the recesses, which occurs in connection with chronic otitis media. The process bears some resemblance to changes coincident with those described in connection with portions of epithelium implanted in the palm of the hand (p. 1241).

Differentiation between true pearl tumors and desquamative otitis is important on the ground that the former demand *radical removal*, together with the bone, while the latter often heals spontaneously. The true pearl tumor is also very frequently the seat of inflammatory processes which extend into the skull and give rise to serious intracranial complications.

Carcinoma and sarcoma do not present any regional pecularities. See Charazac,[19] Körner,[9] and Nos. 6, 7, 8, and 11 of bibliography.

The Operative Opening of the Hollow Spaces of the Mastoid Processes. — The mastoid cells and the antrum are opened (*antrumotomy*) for the purpose of affording egress to inflammatory products, which is often a life saving measure. IT IS NOT SUFFICIENT TO OPEN THE MASTOID CELLS ALONE; THE ANTRUM MUST BE EXPOSED. This is accomplished with the chisel, gouge, and mallet. However, there is no objection to the use of electrically driven instruments. As a matter of fact, the instrumentarium used is very similar to that described in connection with operations upon bones and joints (Part IV), except with regard to size.

The skin is sectioned five to ten millimeters behind the ear, in a curved line down to the lip of the mastoid process; additional room may be obtained by carrying a second incision transversely backward just below the linea temporalis, through which the sinus becomes readily accessible. After the hollow spaces are opened, the wound is lightly tamponed with iodoform gauze. Granulations make their appearance in a few days and healing progresses rapidly, providing the wound is not *too firmly tamponed* — a practice, perhaps, too commonly indulged in by otologists. Small portions of necrosed bone are frequently expelled during the healing, which requires about five or six weeks.

Exposure of all the air spaces of the middle ear (the so-called radical operation of chronic otitis media and cholesteatoma) is destined to afford relief in cases of obstinate middle ear disease which threatens to cause sinus complications. The exposure is made in the manner stated above, but is perhaps somewhat wider in extent, so as to make the posterior wall of the bony auditory canal accessible. The

soft parts are elevated with the elevatorium. Once the antrum is opened, orientation is simple and the entire medial wall of the tympanic cavity may be visualized. At this time the canal of the facial nerve must be identified and its integrity carefully preserved; at the same time, invasion of the horizontal semicircular canal must be avoided. After removing the posterior wall of the tympanic cavity, the drum (if present), the granulations filling the cavity, and the remains of the ossicles come into view. The tympanic cavity is

FIG. 761.— EXPOSURE IN COMPLETED RADICAL MASTOID OPERATION (Dench).

thoroughly "excavated" by means of the bone spoon; the stapes should be preserved, if possible (Stacke,[20] Voss[21]).

The operation is often followed by the presence of an unsightly opening in the bone, for the closure of which a number of plastic operations have been devised. These do not differ in principle from those described in connection with plastic surgery (p. 144). The "blood clot" operation (Blake,[22] Reik[23]) has a limited field of usefulness.

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PART XI

SURGERY OF THE FACE

CHAPTER I

CONGENITAL MALFORMATIONS

Development of the Face.— The clefts occurring in connection with the development of the face are a key to an understanding of the malformations ascribable to disturbances in this connection in early fetal life. The normal formation of the face, as far as malformations are concerned, occur as follows:

As the frontal process growing downward approaches the rudimentary first brachial arch (mandibular arch), the processus maxillares approach from both sides between the submaxillary and frontal processes. This frontal process is divided by the two nasal grooves into a central part (processus nasalis medius) and two lateral parts (processus nasalis lateralis). Two lateral processes (processus globulares), divided by an incisure, project outward from the process.

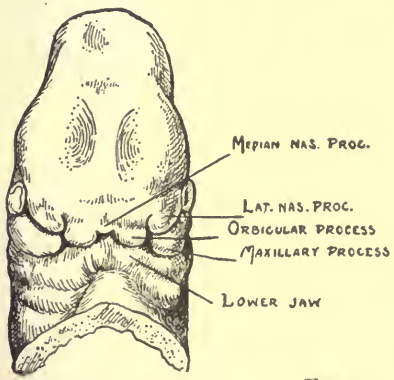

FIG. 762.— FACE OF AN EMBRYO THIRTY TO FORTY DAYS OLD.

The coalescence of the processes and clefts may be represented in a simple manner, if one considers the central nasal process as bounded laterally by two Y-shaped systems of clefts (Merkel[1]). The lower single limb of the Y extends into the oral cavity between the superior maxillary process and the processus globularis, the latter being a portion of the processus nasalis medialis of the two upper limbs; the median passes between the middle and lateral processes toward the nasal fossa, and the outer between the lateral nasal process and maxillary process toward the lower circumference of the rudimentary eye. The cleft of the mouth extends above the lower jaw backward to its lateral origin. The upper lip, together with the corresponding portion

of the jaw, is formed by the coalescence of the middle nasal process and the superior maxillary process. According to Albrecht,[2] the processus nasalis lateralis is also concerned in the formation of these structures, as it gives rise to the outer maxillary process with the corresponding part of the lip and the lateral incisor tooth. The so-called philtrum, the central portion of the upper lip, originates from the central nasal process, while the external is concerned in the development of the ala nasi, and the nostril represents the remains of the nasal groove. The two globular processes unite with each other by means of their median borders.

From the complicated system of clefts between the processes two varieties of malformations occur. Either coalescence of the borders of the cleft is entirely or partially deficient and a rudimentary development or an excessive tissue formation is present, or the coalescence goes beyond the normal and atresia occurs.

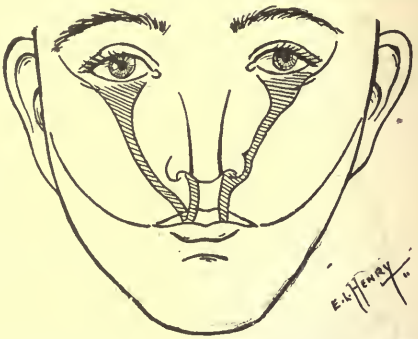

Fig. 763.— Scheme of Facial Clefts (after Merkel).

The *causation* of congenital malformation of this sort is not clear. Whatever influences there are must be operative in the first six weeks of fetal life. The influence of heredity is very probable. Haymann[3] claims that this is shown in twenty per cent of the cases of harelip. It is also probable that amniotic adhesions and bands have a causative influence (Ziegler[4]). See also Lannelongue,[5] Trendelenburg,[6] v. Winckel.[7]

Individual Cleft Formation.— Merkel's[1] schematic representation, in which, however, only the most important forms are shown, gives a general survey of the embryonal clefts (Fig. 763).

Clefts of the Upper Lip.— Clefts of the upper lip may be *median* or *lateral.*

The *median cleft* is rare. It is caused by a failure of coalescence of the median side of the two globular processes and corresponds to the split in the lip of the rabbit. The lightest grade of the deformity is characterized by a slight incurving of the edge of the lip. However, the cleft often extends into the nares and is not infrequently associated with cleft palate (Fig. 772). See Witzel,[8] Bougon et Derocque,[9] Broca.[10]

The *lateral cleft* (also called *labium liporinum*) is the usual one. This form of cleft may be *single, double,* and may be *complicated by cleft palate* in varying degrees. A single lateral cleft may present a very slight degree of *incomplete* incurving of the vermilion border of the lip.

As a rule the lateral cleft appears in the form of an *incomplete defect* located at the side of the philtrum with its apex directed toward the nostril, which it approaches more or less closely, so that only a small bridge of skin separates it from the latter (Fig. 767). The defect is always edged with mucosa and is most widely separated at the line of the oral rim of the lip. In the *complete lateral cleft* the defect extends into the nostril, which is wider than normal and is devoid of a posterior edge; the ala is drawn outward and merges into the outer edge of the cleft. Occasionally a complete cleft is bridged at its center with a small sector of mucous membrane.

Fig. 764. — Double Harelip with Prominent Intermaxillary Bone.

In *double harelip* (Fig. 764) one cleft is usually complete, the other incomplete; as a rule the alveolar arch is separated only on the one side. The philtrum, located between the clefts, is square, triangular, or elliptical in shape.

Most of the cases of complete single or double harelip are associated with clefts of the superior maxilla which are continuous with the clefts of the lips. Of these the severest form is represented by a *complete lip-palate defect* due to failure of coalescence of the edges of the entire frontal process with its neighboring tissues.

Extension of the labial defect into the maxilla and on into the palate is always attended with great deformity of the external parts. When the complete lip cleft (single or double) connects with a lateral maxillary fissure, the intermaxillary bone is likely to be displaced to one side; as the vomer is unrestrained, it grows forward, so that the obliquity of the intermaxillary bone protrudes far beyond the alveolar arch. In this way the point of the nose and the septum are also pushed to one side, while the wing of the nose on that side is broad and flat and stretched over the cleft. The protrusion of the intermaxillary bone is especially marked in double lip-palate cleft. In these cases the defect in the lip is very wide, the vermilion edge is narrow, the nose flat, and the septum and the philtrum are underdeveloped, or the

latter is represented by only a small skin tab. The defect in the alveolar process varies in width from a small superficial fissure to a separation of from 1 to 2 cm. in breadth, extending into the palate. The teeth of the intermaxillary segment are almost always rudimentary and vary in number. In some instances the philtrum and the intermaxillary bone are entirely absent. See Nos. 6 and 9 of bibliography.

Clefts of the Nose.— Clefts of the nose are rare and are the outcome of failure of coalescence of the head segments of fetal life. The *median nasal cleft, dog nose* of Trendelenburg[6] and Witzel,[8] is the most common form of the deformity, and is often associated with malformations of the brain and skull. The nose is separated into two symmetrical halves by a broad median fissure, extending downward between the nostrils, which are separated by the vomer and the septum. Farther down, the cleft extends as a median separation of the upper lip and often involves the alveolar arch and the palate. Beyond this, the *eyes, the frontal processes of the superior maxillae, and the wings of the nose* are widely separated; the nasal bones may be absent. The halves of the nose appear as two cartilaginous tubes, the median walls of which are not in contact. In less severe cases the clefts of the palate, the lips, and the intermaxillary bones may be absent; the nose is flattened, the nasal cartilages form the upper wings of a Y, and the rest of the malformation may be restricted to a shallow indenture in the dorsum of the nose. Congenital nasal fistulae and dermoids are to be regarded as remnants of malformations of this class. The embryology of the malformation is somewhat complex. See Witzel,[8] Lexer,[11] Kredel.[12]

Fig. 765.— Median Nasal Cleft (Dog Nose).

The *lateral nasal cleft* is still more infrequent; it is easily mistaken for the malformation coincident with oblique facial cleft (Broca,[10] Frangenheim,[13] Nash[14]).

Oblique Facial Clefts.— Oblique facial clefts (meloschisis) present great varieties. They often coëxist with cerebral anomalies, in which event the infant (fortunately) does not survive. In a general way

the cleft extends from the upper lip to the eye and involves either the soft parts, or the soft parts and the bones. The malformation of course varies widely in extent and is often double. Only those of minor degree are susceptible to correction by surgical efforts at relief. See Broca,[10] Frangenheim,[13] Nash,[14] Morian.[15]

Transverse Facial Clefts.— Transverse facial clefts (macrostomia) are not so rare as the oblique. They represent a failure of coalescence of the superior and inferior maxillary processes. The cleft is edged with mucosa and often extends back to the anterior edge of the masseter muscle; it may be bilateral. The lesser degrees of the deformities are restricted to a large mouth, although even in these instances a band of scar tissue may reach to the ear. The typical fissure ends in front of the tragus, while others extend through the zygoma to the temporal region. Atypical clefts are caused by amniotic bands and may occur over the superior or inferior aspects of the cheek (Chavane[16]).

Fig. 766.— Oblique and Transverse Facial Clefts.

Median Clefts of the Lower Lip.— Median clefts of the lower lip and the inferior maxilla are not common. They are caused by faulty coalescence of the arch of the lower jaw and represent a simple median fissure or extend through the entire bone. The tongue and the floor of the mouth may exhibit simultaneous median clefts of varying degree (Salzer[17]).

The Operative Relief of Cleft Formations.— Operations for the relief of cleft of the upper lip are legion. A few, illustrating the principles involved, will be discussed here. The older methods of apposing the refreshed edges have been abandoned in favor of the silk or silkworm gut suture. The apposition must be exact and tension must be avoided by the introduction of two or three retention sutures. A simple method of dealing with a slight degree of *uncomplicated* harelip (attributed to Nélaton) is shown in Fig. 767. Wide defects may be treated by the method of Mirault,[8] which is variously depicted in the literature. Fig. 768 shows the method as the writer understands it

and is especially distinctive, as it crosses the site of the defect with mucosa, which is now normally placed. This, after all, is the keynote

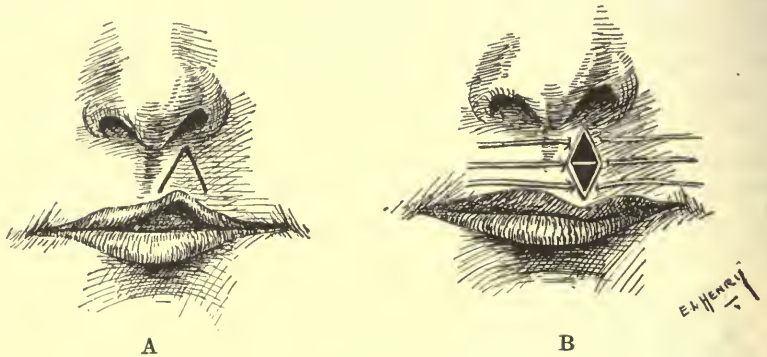

FIG. 767.— NÉLATON'S METHOD OF DEALING WITH SIMPLE HARELIP. *A*, INCISION; *B*, SUTURE.

of all plastic lip operations. See also methods of Hagedorn,[19] and résumé by Lexer.[20]

The operative relief of double harelip, unless complicated by the protrusion of the intermaxillary bone, does not differ in principle

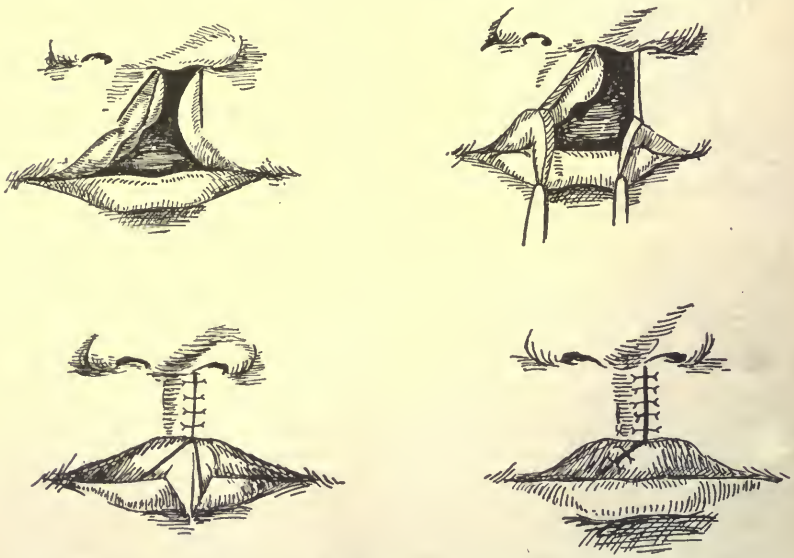

FIG. 768.— THE STEPS OF MIRAULT'S OPERATION FOR HARELIP.

from that already described. Here again, Mirault's[18] old method (Fig. 769) best meets the indications in most cases. Lexer[20] in his presentation depicts a number of methods and also describes in detail his own procedure devised to obviate "inrolling" of the repaired lip.

In cases of *double harelip with protrusion of the maxillary bone,* a

FIG. 769.— MIRAULT'S METHOD OF CORRECTING DOUBLE HARELIP.

number of methods are employed. The older one of resecting the protruding bone has been abandoned. Of the various methods of overcoming the deformity, *subperiosteal section of the vomer and septum* (v. Bardeleben,[21] Delore[22]) is the one most generally practiced. After the mucoperiosteal covering of the lower edge of the vomer is incised longitudinally, it is freed on both sides by means of the periosteal elevator and the vomer is divided with bone cutting forceps. Light backward pressure replaces the intermaxillary bone and causes the two bony segments of the divided vomer to overlap (Fig. 770). The overriding vomer is held in place by a catgut suture and the soft parts are closed over its edge. See Krause-Heymann.[23] Partsch[24] resects a wedge of the vomer with an especially constructed forceps. Lexer,[20] in his summary, states the methods of choice to be as follows:

FIG. 770.— INTERMAXILLARY BONE REPLACED.

Overlapping sectioned vomer held in place with a single suture.

For single harelip, (1) complete and incomplete, use Mirault's[18] method; (2) for small incurving of vermilion border, use the method of Langenbeck as described by Wolff,[25] which is very similar to that shown in Fig. 768. For double harelip use Mirault's[18] method (Fig. 769) or, when the philtrum is small, use that of Hagedorn;[19] for

reposition of the premaxilla, use the method of v. Bardeleben[21] (Fig. 770).

The *after treatment* is important. The nostrils must be kept free from secretions so as to enable the child to breathe while nursing. The application of the protective dressing is attended with difficulties upon which much ingenuity has been exercised. As a matter of fact, healing takes place with the least disturbance when the wound is simply kept covered with sterile zinc ointment; the child's hands are tied. The crust formed by the ointment and the secretions is easily removed. The sutures are taken out on the fifth day; light narcosis is advisable.

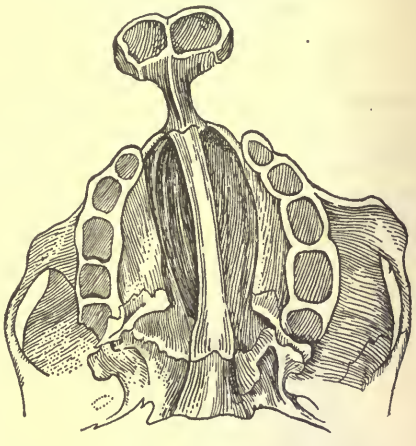

Fig. 771.—Double Cleft Palate.

Operative correction of the other facial clefts is not usually successful. What has been attained has been the outcome of the application of the general principles of plastic surgery. See Lexer.[20] Of the *abnormalities in the areas of the fetal clefts and furrows,* only a few possess surgical importance. These are cases of *complete atresia of the eyelids,* the *nostrils,* and the *mouth.* To these may be added an abnormal coalescence of the mouth cleft, which produces so-called *microstomia.* It is easily conceivable that all kinds of malformations are liable to occur in the fusion of the various head segments, which include those merely offensive from an artistic standpoint to those representing unsightly deformities.

In the *region of the eye,* malformations cause peculiarities in the formation of the eyelids which at times involve the lachrymal sac Schanz[26]). Dermoids and fistulæ in this region are also ascribable to congenital causes.

Fistulæ of the upper lip, the *nose,* and the *lower lip* are also the result of faulty coalescence of the facial clefts. Occasionally, the *transverse cheek cleft,* and more frequently the *oblique* are the seat of fistulæ (Trendelenburg[6]).

The *correction of malformations* of this sort is carried out along

the lines of plastic surgery in general. Fistulæ must be widely excised and the residual opening carefully sutured.

So-called *double lip*, in the form of a thick protrusion of mucosa, appears mostly at the upper lip and is especially noticeable during laughter. The condition is readily corrected by excision.

Cleft Palate (Uranoschisma).— As in clefts of the face, failure of coalescence of the head segments in fetal life gives rise to congenital deformities of the roof of the mouth. In most cases these complicate cleft formation in the upper lip and appear as defects of the hard palate of varied form and extent. Their formation is explained in a similar manner to that of the abnormal facial clefts. The *failure of coalescence* of the frontal and maxillary processes is due to various causes, which are very similar to those already discussed, and, in some instances, is no doubt due to the *traction of amniotic bands* (Dursy,[27] Fernet,[28] Draudt[29]).

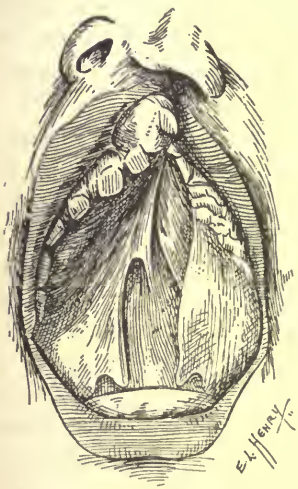

FIG. 772.—LEFT SIDED HARE-LIP AND CLEFT PALATE.

Marked displacement of intermaxillary bone.

Union of the palatine plates with one another and the vomer may not take place at all, or may occur only on one side, or in a few areas and remain open in the others. Thus, clefts may be divided into *complete and incomplete. Complete cleft palate* (*uranoschisma*), as far as the hard palate is concerned, may be single or double (uranoschisma unilateral or bilateral), while the incomplete cleft (unanocoloboma) may be anterior or posterior. The amount of separation varies with the degree of development of the palatine processes.

Double cleft palate is as a rule medial at its posterior end and divides to either side of a protruding intermaxillary bone (Fig. 771). This severe form is called "wolf jaw" and is very difficult to correct. According to v. Langenbeck,[30] this form of the malformation is likely to unite with the vomer on one side (usually the right) before the second year of life. The intermaxillary may be united on one side.

Unilateral cleft palate is the form most often seen (left side). In these cases the cleft is in the median line in the soft palate and is continued up into the left nostril through the jaw and lip (Fig. 772).

If there is a double harelip present, only one of the clefts communicates with the lateral cleft palate. On the cleft side the palatine arch is very rudimentary, i. e., the superior maxillary is underdeveloped.

Incomplete cleft palate (uranocoloboma) of the hard palate also varies in degree. At times, it is associated with single or double harelip and with a median cleft of the soft palate. The anterior defect represents a continuation of the cleft in the alveolar process; to this, occasionally, a cleft in the soft palate is also added while the greater part of the hard palate is closed. A posterior defect of the hard palate is often associated with a cleft in the soft palate. The rarest form of the defect is seen when the palatine processes of the superior maxilla fail to unite and the horizontal plates of the palatine bones and the velum are fully developed. In cases of this sort the defect is located in the median line.

Clefts of the soft palate may be present alone, in which instance they are characterized by a simple incurving of the uvula, or the defect extends to the posterior edge of the horizontal plates of the palatine bones. As a rule, however, the cleft is associated with a greater or lesser defect of the bony palate.

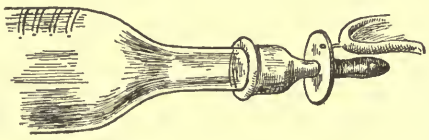

FIG. 773.—CLEFT PALATE OBTURATOR FOR INFANTS (Warnekros).

Intra-uterine healing of clefts of the palate is shown by a slight separation of the bones, which is covered with thin mucosa. Usually, the velum is abnormally short and imparts a nasal character to the speech (Küster[31]).

The *disturbances* consequent to cleft palate relate (1) to interference with *nursing and deglutition*, and, later, with *speech;* and (2) to *inflammation of the mucous membranes*. The latter is expressed in all forms of inflammatory processes involving the nasopharynx and the eustachian tube with its dire consequences to the auditory apparatus. The inability, in children, to take food and the overflow of milk through the nostrils is a distressing contingency — one which often causes the death of the infant (pneumonitis, bronchitis, etc.). Temporary assistance in feeding may be acorded by the device of Warnekros[32] (Fig. 773). In later life the disturbances of speech are a great affliction. See Gutzmann.[33]

The operative measures of relief in cleft palate are based on the early work of v. Graefe,[34] 1816, and v. Langenbeck,[35] 1861. Between these periods a number of surgeons devoted much effort to improving the

technic of the procedure, notably Dieffenbach,[36] Fergusson,[37] Lannelongue,[5] and Roux.[38] The corrective proeedures are ealled *staphylorrhaphy* and *uranoplasty* or, when both the hard and soft palate are defeetive, the term *urano-staphyloplasty* is used. The operation in all cases should be preceded by removal of hypertrophied pharyngeal tonsils. Chloroform narcosis is used by most surgeons. The flow of blood into the traehea is obviated by placing the patient in *Rose's posi-*

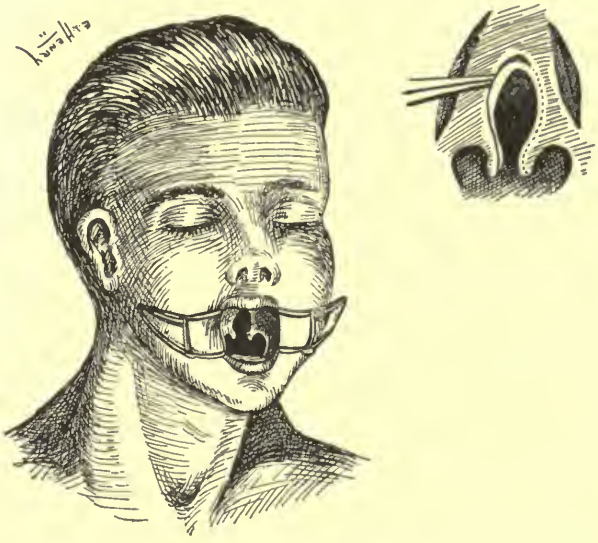

FIG. 774.— POSITION OF HEAD IN CLEFT PALATE OPERATIONS.

Whitehead mouth gag in position. Insert shows how the edges of the defect are refreshed.

tion (Fig. 774), supplemented by the use of a suction apparatus. The mouth is held open with a Whitehead gag (Fig. 774).

The technic of the proeedure is divided into four acts.

1. *Refreshing the edges of the defect* is aeeomplished by picking up the tip of the soft palate with a long tenaculum and, with a thin sharp knife, denuding the edges of the separated soft palate. The edges of the hard palate are trimmed off with a stiff eartilage knife.

2. The *lateral incisions through the involucrum palatum,* which make loosening and medial displacement of the flaps possible, are earried through the mueosa and the periosteum. They begin near the hamulus, are carried close to the teeth, and end in front between the

lateral incisors. The flaps must not be too narrow in front; in this way injury to the palatine arteries is avoided. Bleeding is usually inconsiderable. As soon as the first incision is made, it is tamponed with iodoform gauze.

3. *Elevation of the mucoperiosteal flaps* is the most important step in the operation. It is best accomplished from the lateral incisions; the tampon is removed and the flap raised by means of an elevator. When the flap is raised, the *soft palate is relaxed* by inserting a small chisel into the posterior aspect of the lateral wound (obliquely inward and upward) toward the hamulus and fracturing it. The lateral tampons are then replaced.

4. *Exact apposition of the denuded surfaces* is the final step of the operation. This is readily attainable by means of an appropriately fashioned needle holder. Silk is usually used for the purpose. The suturing is begun from behind. Some surgeons use silver wire and others use silkworm gut, which may be fastened with shot. When only the soft palate is defective, relaxation incisions may not be necessary. *Small defects* in the bony palate are treated in the same way as the larger ones. *In complete double maxillary-palatine clefts* the harelip is corrected first and, after this is healed, the defect in the palate is closed.

In the *after treatment,* sterile fluid nourishment is administered and the mouth is frequently cleansed with a mild sterile disinfectant solution. *At the end of a week,* the tampons and sutures are removed. The sterile diet and painstaking cleanliness are maintained for another week. While the operation is followed by great anatomical improvement, speech is never normal. However, this may be astonishingly improved by systematic "speech and breathing exercises" (Gutzmann[33]).

In very severe form of cleft palate, the operation described above does not suffice. In these cases a number of methods are employed. For instance, Lane[39] turns down a flap from the side of the roof of the mouth and sews it into the previously denuded defect; Moskowicz[40] uses a flap from either side of the defect and, after inverting them, sutures them over the defect; v. Eiselsberg[41] mobilizes the vomer and turns it down into the cleft. See also Lexer.[20]

When the malformation does not interfere with nutrition and delay is permissible, it may be advisable to massage the separated alveolar processes with the view of reducing the width of the cleft before operating. Brophy[42] denudes the edges of the bony defect, forces the

two maxillae together, and holds them apposed by means of silver wires passed through and through the bones just above the alveolar processes. The wires are twisted over lead plates and are kept in place for three weeks. The soft palate is repaired later.

The supplementary use of a prosthesis improves the character of speech very much. All of these are based on the principle of preventing air from entering the nasopharynx. The principle was worked out by Sürsen,[43] after whose original apparatus all subsequent ones are modeled. See also Schiltsky,[44] Warnekros,[32] Lexer.[20]

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CHAPTER II

INJURIES OF THE FACE

Contusions of the face, even slight ones, are attended with considerable effusion of blood, except where the skin is closely attached to the bone, as over the zygoma, the chin, and the bridge of the nose. *Hematomata* of the eyelids and cheeks are particularly menacing in appearance. However, sloughing rarely ensues. *Lacerated wounds* are produced by blunt, tearing force; avulsion of the soft parts is rare, although tearing of the nostrils and mouth (by a cane, an umbrella, a fence picket, etc.) is not so uncommon. The application of *great force* (such as an explosion, grenade fragment, bird shot, etc.) is followed by extensive laceration of the soft parts and grave injury to the bone.

Aside from the very extensive injuries, wounds of the face *heal more rapidly* than those located elsewhere in the body and are comparatively rarely infected. This is explained by the great vascularity of the face, and by the free bleeding which mechanically cleanses the wound surfaces. However, a policy of complacency in the latter regard is to be deprecated, for, if infection does develop, it is likely to be attended with grave consequences. For this reason contused and lacerated wounds should be carefully cleansed, and, when sutured, ample provision for drainage must be made. The necessity for apposition of the wound surfaces is especially urgent in injuries involving the eyelids and the lips. The *sutures must be widely separated from one another.* The treatment of this class of cases is extensively discussed elsewhere (Part I, chap. iii).

Incised wounds of the face are often complicated by involvement of the nasal passages and the mouth. Loss of substance by cutting instruments is particularly distressing in this situation (traumatic amputation of the nose, lips, etc.). Wounds of this sort are very likely to be attended with division of *large vessels* and *nerves,* including Stenson's duct. As already stated, wounds of the face bleed very profusely, especially those involving the anterior maxillary, the superficial and the deep temporal arteries.

Deep, incised wounds of the cheek and parotid region often divide the branches of the *facial nerve* with consequent paralysis of the muscles. The larger branches of the nerve are easily repaired by suture (see nerve suture, p. 579). When the nerve is not primarily sutured, secondary repair may be practiced. If this fails, anastomosis of the peripheral stump with the spinal accessory or hypoglossal nerves may be followed by restoration of function (Ballance and Stewart,[1] Cushing,[2] Frazier and Spiller,[3] Körte[4]). However, muscular action is attended with so-called associated movements of the shoulder and tongue which mar the benefits derived from the procedure. In this class of cases, *muscleplasty* as practiced by Lexer[5] gives gratifying results (see also Nordmann[6]). For the purpose, Lexer[5] makes an incision over the anterior temporal region, exposing the fibers of the temporal muscle, from which he separates a goodly bundle of fibers (Fig. 775). After the skin is tunneled, the fibers are drawn forward by means of two silk ligatures and the latter knotted close to the eyelid. A similar flap is fashioned from the anterior edge of the masseter muscle and fastened in the same manner in the nasolabial fold (Fig. 775).

Fig. 775.—Muscleplasty in Facial Paralysis (Lexer).

Division of the branches of the trifacial is rarely followed by anesthesia; the nerve usually regenerates rapidly. Adherence of a branch of the nerve to a scar may be the cause of neuralgia. In these cases a small neuroma usually develops in the scar or close to it, and when this is removed the pain disappears.

Wounds of the cheek, especially those penetrating into the mouth, are likely to involve the duct of the parotid gland, and, occasionally, an external salivary fistula is the result. The condition is easily corrected by splitting the duct and turning it into the mouth through a stab wound in the mucosa (p. 1629). Injuries of the muscles heal without loss of function, except when the *levator palpebrarum is severed;* in this event, ptosis develops. It is necessary to expose the muscle and suture it (Weber[7]).

Incised wounds of the face are especially responsive to careful *repair by suture.* If bleeding is thoroughly arrested and the edges of the wound carefully apposed, primary union almost always follows. *Portions of the face that are completely severed* (point of the nose, external ear) should be carefully sutured into place; in a number of instances a favorable result is thus obtained (v. Bergmann[8]).

Gunshot wounds of the face usually involve the bone. They do not present a special problem, except that which is peculiar to their location. Suicidal wounds, when the revolver or other firearm is held in the closed mouth, are likely to be extensive — the result of the explosive action of the discharge. Wounds of this sort are important because of the *complications,* i. e., *injuries of the brain, interference with respiration — the result of injury of the mouth* and involvement of the *large vessels.* The injury to the mouth and tongue may be attended with severe primary hemorrhage from the branches of the lingual and internal maxillary arteries, with secondary bleeding or with inflammatory swelling of the soft parts, either of which may demand *immediate tracheotomy.* Arrest of the bleeding may be attended with considerable difficulties. It is, of course, desirable that the divided vessels be ligated in the contiguity of the wound. It may, however, be necessary to *tie the external carotid in its continuity* in order to control the bleeding.

Gunshot wounds of the face are *more likely to be infected* than are other classes of injuries. This is due, in part, to the fact that comminution of bone is always attended with great trauma and more or less extensive bloody infiltration of the soft parts, and also because many of the wounds communicate with the mouth, the nose, and accessory cavities. All of these conditions favor the occurrence of *secondary hemorrhage.* The latter is particularly disquieting, as attempts to ligate the bleeding points only spread the infection, and, therefore, it becomes necessary to tie the afferent vessel or vessels in their continuity (lingual, temporal, external carotid). A certain number of gunshot wounds of the face are followed by the development of *traumatic aneurisms.*

Injuries of the eye by birdshot, with total destruction of the eyeball and lids, are not uncommon. The most severe injuries to the contents of the orbital cavity occur when a projectile enters it through the temporal region.

Injury of the facial and trifacial nerves by projectiles do not differ

from those described above in connection with incised wounds of these nerves.

The *treatment* of gunshot wounds of the face, other than that just discussed, is treated in the general part of this work (p. 908).

Freezing and burns of the face are especially important because of the distressing deformations sequential to healing. The *eyelids* (*ectropium*), the *nostril,* and the *mouth* are especially likely to be the sites of cicatricial deformations. These may, to a considerable extent, be avoided by the prevention of suppuration and the *early employment of skin grafting.* In this class of cases the *Thiersch method* (p. 20) is particularly useful.

For Acute and Chronic Infections *see* general part.

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CHAPTER III

PLASTIC SURGERY OF THE FACE

Rhinoplasty.— Replacement of the nose with a single layer of skin is always followed by shrinkage of the flap. This was in a measure overcome by Dieffenbach,[1] who used a double flap, i. e., one with skin on both sides. The introduction of artificial bridges failed. An important step forward was taken by v. Langenbeck,[2] who is responsible for so-called *periosteal rhinoplasty,* in which a flap consisting of skin and periosteum was taken from the forehead. Later, v. Bardeleben[3] turned a flap of this sort down from the forehead and covered it with skin flaps derived from the cheeks, which he slid over the raw surface of the former. In 1861 Ollier[4] used a bone flap from the nasal process of the superior maxilla, which he slid under the periosteal flap of v. Langenbeck. This opened the way to *osteoplastic repair of the nose.*

The plastic operations on the nose are employed for the relief of *saddle nose* and for *complete and incomplete rhinoplasty.*

The *operative relief of saddle nose* is employed when the point, the alae and the septum are preserved. The deformity is usually the outcome of destruction of tissue due to *lues,* though it may result from *trauma, tuberculosis and acute osteitis.* The *operative method of Koenig*[5] is as follows:

A transverse incision through the deepest portion of the depression mobilizes the soft parts so that they can be pulled down to their normal position. The resulting defect, which extends into the nasal cavities, is covered by means of a flap from the forehead. This flap is a prolongation of the nasal bridge with its base at the root of the nose; it is cut out of the forehead so that it is six to seven centimeters long and only one centimeter in width (Fig. 776). After the skin and periosteum have been incised, the cortical portion of the bone is chiseled through along the cut in the periosteum. The flap of skin and bone thus outlined is now separated from the diploë from above downward by means of a flat thin chisel; the flap is fractured at its pedicle, inverted and sutured into the defect. Thus the shell of bone

forms the external and the skin the internal surface of the nose. The lower edge of the inverted flap is sutured to the mobilized lower soft parts so that the skin of the latter overlaps the former. The skin flap, which is taken from the forehead in the usual manner, is sutured upon the raw surface of the first flap and forms the external skin of the newly formed bridge of the nose (Fig. 776 B and C).

The small "skin tab" left at the root of the nose is trimmed after several months. After this, a number of "adaptation operations"

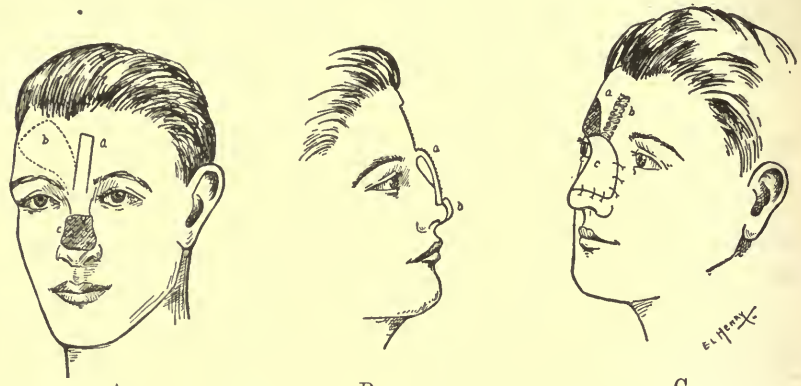

FIG. 776.— KOENIG'S SADDLE NOSE OPERATION.

A, *a*, Bridge of nose flap (skin and bone); *b*, skin flap from forehead; *c*, defect formed by transverse incision.

B, *a*, Bridge of nose flap turned down; *b*, held in place by catgut sutures. The skin at *b* remains free for union with forehead flap.

C, *a*, *b*, Defects in forehead; *c*, skin flap from forehead sutured in place over the bridge of the nose.

are usually necessary in order to fashion the newly made nose in accord with the rest of the face.

A number of modifications of the method have been devised by various surgeons. Of these, that of Israel[6] is worthy of consideration (see bibliography). Transplantation of portions of bone from the tibia or a rib, and the implantation of decalcified bone, are also practiced with more or less success.

In simple saddle nose, in which it is *not necessary to open the nasal cavities*, a simple procedure often suffices. A transverse incision is made over the root of the nose down to the periosteum, the soft parts of the nasal bridge are elevated with a periosteal elevator, and a section of bone of suitable dimensions (from the tibia or a rib), together with its periosteum, is introduced. In this class of cases the method gives

excellent results, provided the cavity of the nose has not been entered during the manipulations (v. Mangoldt,[7] Albee[8]).

In the severest forms of saddle nose, with much shrinkage of the soft parts, Lexer[9] splits the nose longitudinally and uses the skin as an interior lining for the new nose.

Complete rhinoplasty is necessary when the nose is entirely lost (carcinoma, syphilis, trauma, etc.). The available methods are designated as the French, Indian and Italian.

The *French method* (Nélaton,[10] Serre[11]) is used when the bony framework of the nose is preserved. The defect is closed by means of two lozenge shaped flaps taken from the cheeks. Today the method

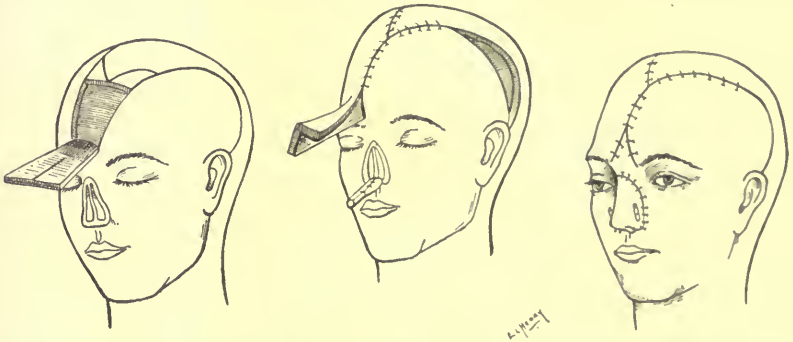

FIG. 777.—SCHIMMELBUSCH'S RHINOPLASTY OPERATION.

a, Bone flap from forehead turned down.
b, Defect in forehead closed by transplanted curved scalp flap.
c, Result of plastic operation.

is used only to cover with skin an inverted osteoplastic flap taken from elsewhere. Of the other procedures, *only those the fundamental principle of which consists in the formation of a flap of skin and bone retain practical value.* Of these, the method of Koenig[5] is modified so as to meet the indications in complete rhinoplasty (Rotter,[12] Schimmelbusch[13]). Schimmelbusch[13] forms a flap from the forehead much like that described in connection with saddle nose (Fig. 777), except that it is made larger. The base measures from 2 to 3 cm. between the eyebrows and the upper end from 7 to 9 cm. close to the hair line. A thin plate of bone is elevated with the flap by means of a broad, thin chisel. The flap is then wrapped in iodoform gauze and the plastic closure of the forehead carried out as shown in Fig. 777. The raw surfaces of the flap may now be covered with skin or, which is

better, the formation of granulations may be awaited (four to eight weeks) and the grafting be done then. After the raw surface is covered with skin it is implanted into the nasal defect. The bone plate is sawed in the middle and angled like the roof of a house and the flap is twisted so that the skin is turned outward. By refreshing the defect the bony edge of the pyriform aperture is exposed and the

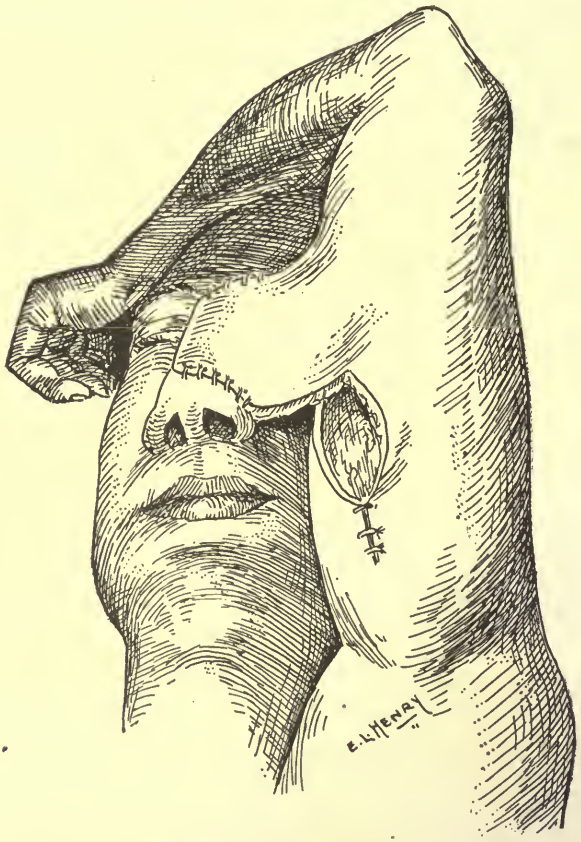

FIG. 778.— IMPLANTATION OF FLAP UPON THE NOSE.

nasal flap, the sides of which have also been refreshed, is placed accurately upon it. If it is necessary to construct a septum, this can be taken from the skin edge of the pyriform aperture, after it has been refreshed, in the form of two thin skin flaps (Fig. 777). In three weeks the pedicle of the frontal flap is cut through.

The operation is not without its technical difficulties and has certain

objectionable features. Many of the latter have been overcome by Lexer,[9] who employs the principle of Koenig[5] but modifies its technic in a manner which gives excellent results. A description of the procedure is too elaborate to reproduce here. See bibliography.

The *Italian method of rhinoplasty,* which employs flaps from the arm, is rarely used at the present time, except when skin is utilized to cover a previously reconstructed bony defect (Fig. 778).

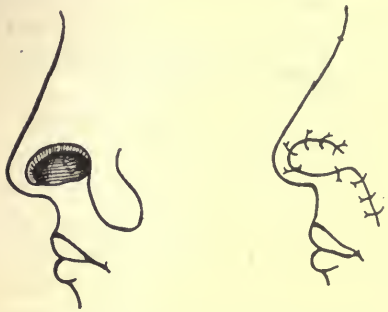

Fig. 779.— Repair of Defect in Wing of Nose.

Incomplete rhinoplasty is employed for the purpose of *replacing portions of the nose,* such as one half of the nose, an ala, tip or septum. Defects of this sort are caused by *lupus, lues, carcinoma and trauma.*

A *defective wing of a nostril alone* is readily closed by the use of a pedunculated flap taken from the contiguous nasolabial fold (Fig. 779).

Cheiloplasty.— Plastic operations on the lips are destined to correct defects or to improve deformations. All or a part of a lip may be destroyed as the result of *noma, gangrene,* and in the removal of *new growths.* Among the latter, *carcinoma of the lower lip* often demands extensive removal of substance. Deformation of the lips is frequently sequential to cicatrization following *tuberculosis, lues, burns,* etc. Plastic repair of the lip goes back to early history. However, modern cheiloplasty is based on the work of Dieffenbach,[1] who first employed the entire thickness of the cheek, including the mucosa, for the purpose.

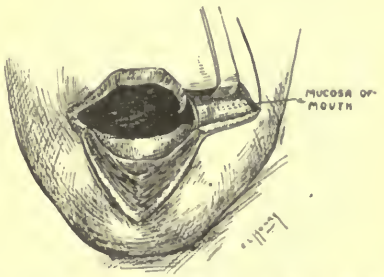

Fig. 780.— Greater Part of Lower Lip Excised, Liberating Incision in Cheek Down to Mucosa of Mouth.

Of the many methods more or less transiently *en vogue, only those which include covering the new lip with mucosa are of practical value.*

In complete or incomplete replacement of the lower lip, as is frequently necessary in connection with operative removal of carcinoma

in this situation, the correctional procedure varies with the amount of normal tissue it is necessary to remove.

After removal of *small, superficial new growths,* the mucosa of the lip may be removed by means of *transverse elliptical incisions* and subsequent suture; on the other hand, *larger processes,* which invade the skin, should be removed by *wedge shaped excision* of the entire thickness of the lip, and the point of the wedge must extend well into the chin. *One half of the lower lip may be removed* in this way and the edge of the defect apposed by suture without any serious subsequent deformity.

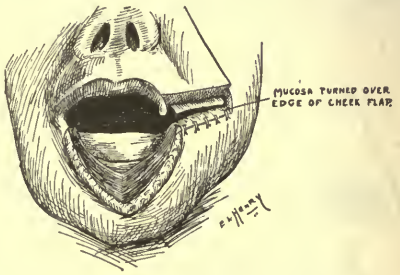

FIG. 781.—PLASTIC FORMATION OF BORDER OF LIP.

Still larger defects are closed by the modified Dieffenbach[1] method as follows: An incision is made from the angle of the mouth in the direction of the external auditory meatus, crossing the masseter muscle to a finger's breadth in front of the ear. The knife divides only the tissues down to the mucosa of the mouth (Fig. 780). A rectangle of mucous membrane, 1 cm. high, is fashioned so as to retain its connection with the lower flap of the cheek (Fig. 781). At the end of the transverse cheek incision an incision is made running obliquely toward the inner corner of the eye (Fig. 781), and the lower flap is mobilized from the alveolar processes until the mucous membrane edges of the defect can be brought together within the mouth and closed with buried catgut sutures. By drawing up these sutures the mucous membrane of the remnant of the lip and the mucous membrane of the cheek flap are approximated. This is followed by approximation of the old and the newly formed lip borders (Fig. 782). The end of the oblique incision in the cheek is sutured to the corner of the wound of the upper lip — and

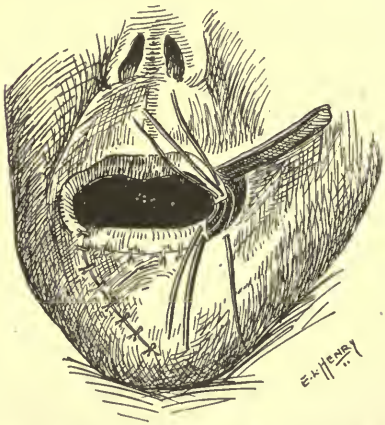

FIG. 782.—COMPLETION OF SUTURE OF LIP; FORMATION OF CORNER OF MOUTH.

after a triangular piece is excised from the redundant cheek tissue, this space is also sutured (Krause-Heymann[14]).

In *complete loss of the lower lip* two flaps are fashioned in the manner indicated in Fig. 783, the mucous membrane of the cheek being utilized in the manner just described (v. Bruns[15]).

As is the case with plastic repair of the nose, that of the lip is attainable by a number of methods too numerous to relate here. All of these, however, are based on the principles of the methods described above. See also Abbe,[16] and especially Lexer.[9]

Stomatoplasty.— *Ectropium of the lip* is corrected in the same way as is the similar deformity of the eyelid; a V-shaped incision is sutured in the form of a Y. In severe cases the scar must be excised and the lip replaced by pedunculated flaps.

In contracture of the oral orifice the mouth is enlarged by means of lateral incisions and the mucosa utilized in the manner described in

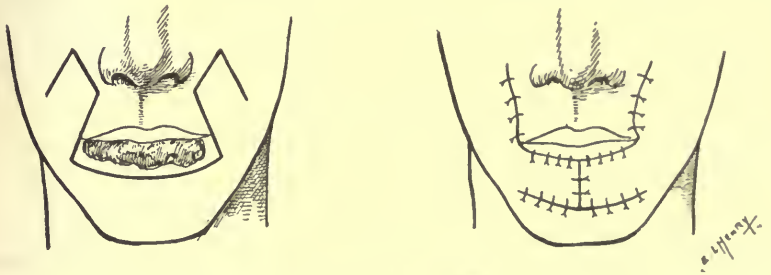

FIG. 783.— RESTORATION OF LOWER LIP (v. Bruns).

connection with defects of the lower lip. For extensive operative measures in this connection see Lexer.[9]

Meloplasty.— The plastic repair of the cheeks is often necessary in connection with stomatoplasty or cheiloplasty in cases of *defects of the cheek* or in *cicatricial ankylosis of the lower jaw.* In the latter class of cases the mobility of the jaw is restored by removing the cicatricial tissue and replacing it with normal; hence, the CHIEF PRECAUTION IN EVERY PLASTIC OPERATION UPON THE CHEEK IS THE AVOIDANCE OF SHRINKAGE OF THE IMPLANTED FLAP. As in other situations, the implantation of a single layer of skin flap is followed by contraction and interference with function. It is, therefore, necessary to replace the mucosa with one layer of transplant and the skin with another (Gussenbauer[17]). In suitable cases, the mucous membrane contiguous to the defect may be slid or jumped into its buccal aspect and a similar

procedure may be carried out on the face. However, this is not always feasible, especially when the loss of substance is great. In these cases it is necessary to replace the mucosa with one layer of skin

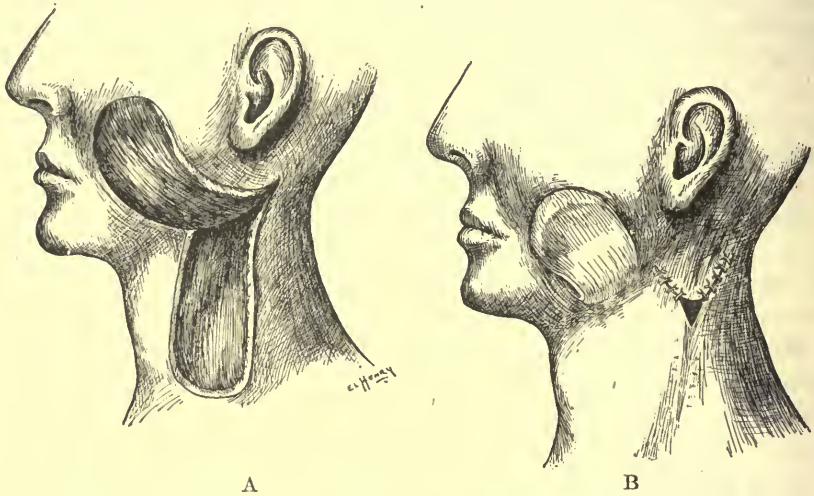

FIG. 784.— PLASTIC REPAIR OF CHEEK.

A, Flap carried to cheek from neck; *B*, folding the flap over, after dividing the pedicle.

and the cheek with another in the manner shown in Fig. 784 A and B (Israel[18]). The pedicle of the flap is severed fourteen days after the primary transplantation and folded on itself as shown in Fig. 784B.

FIG. 785.— BLEPHAROPLASTY (v. Langenbeck).

The fold is trimmed off and sutured fourteen days later. In less extensive loss of substance it is often possible to fashion a mucous membrane flap from the floor of the mouth and the side of the tongue, which is used to close the defect on the inner aspect of the cheek;

the outer may then be closed in the usual way (Lexer[9]). Care must be taken to replace the mucous side of the defect with skin devoid of hair. Stenson's duct and the facial nerve must be shown proper consideration.

Plastic Replacement of Hairy Areas.— For cosmetic reasons at times it is advantageous to utilize flaps covered with hair for purposes of replacement of loss of substance. For the *eyebrows* and *mustache* pedunculated or free flaps may be taken from the scalp. However, the former are far more satisfactory, as the hair follicles of a free implant are exceedingly likely to undergo insular or total atrophy. For the *eyebrow*, a flap may be advantageously taken from the hairy scalp of the temporal region. A *mustache* is readily formed by jumping flaps into a defect of the upper lip from the chin.

FIG. 786.— BLEPHAROPLASTY (Dieffenbach).

Blepharoplasty.— Defects of the eyelids are caused by *trauma*, or by ulcerative processes in connection with *tuberculosis, lues, necrosis following erysipelas or anthrax;* or it may be the outcome of the radical *extirpation of neoplasms.*

In determining the technic of blepharoplastic measures it is important to know whether the *skin alone or the conjunctiva also is destroyed.* In the latter instance, a flap consisting of skin only is always followed by shrinkage of the implant and return of the deformity. Lexer[9] divides the surgical problem into *four operative groups.*

To the first belongs the *Indian method,* in which an ample pedunculated flap for the upper lid is taken from the temporal region and for the lower lid from the cheek. Fig. 785 shows the method used in connection with the lower lid (v. Langenbeek[2]).

The method of *sliding in a direct line* (Dieffenbach[1]) is based on the principles of plastic surgery discussed elsewhere (p. 144); its application to the eyelid is shown in Fig. 786.

When it is necessary to construct an entire new lower lid, an

incision somewhat longer than the base of the defect is carried horizontally outward from the external canthus; a second incision divides the tissues downward, parallel to the outer edge of the defect. A rhomboid shaped flap is thus formed, which is dissected off and slid into the defect. Celsus[19] liberates a flap from either side of the defect in much the same manner as is shown in connection with repair of the lower lip.

The fourth method of repair consists in *transplantation of a free flap* (Wolfe[20]). See Plastic Surgery, p. 144.

When it is also necessary to *replace the mucosa,* the inner aspect of the flap is covered by the methods of Uhthoff[21] and Jordan.[22] The former fashions a triangular flap from the *mucosa of the upper lid,* which he turns into the defect of the lower lid. At the end of the eighth day the flap is severed from its upper attachment and the skin defect is closed in the usual way. Jordan[22] uses a *double skin flap* from the forehead in the same manner as is shown in Israel's method of meloplasty. The implantation of a free transplant of mucous membrane is of doubtful usefulness (Wölfler[23]).

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CHAPTER IV

NEURALGIAS OF THE HEAD

Neuralgia in general has already been discussed (p. 1032). However, the problem in connection with the head possesses some particular features which are taken up at this time. Of the superficial nerves, the *trigeminus* (with the exception, perhaps, of the sciatic) *is most often the seat of neuralgia.* Whether this is due to the variety of functions that this nerve is concerned in or to its peculiar anatomical relationship is not clear. The fact that its branches traverse a number of bony foramina may have a bearing upon this aspect of the solution.

Pain, which after all is the dominant factor in trifacial neuralgia, is always sudden in onset and varies widely in its intensity; however, it is never slight. It is described as stabbing, tearing, boring, and lancinating, and at times is so unbearably severe that the patient commits suicide. In many instances, the pain is paroxysmal and an attack may be induced by relatively slight influences, such as a light touch on the skin, a breath of cold air, muscular movements coincident with laughter, speech, swallowing, chewing, etc., and by psychic influences. A paroxysm may last for only a moment. Sometimes the attacks occur every few minutes for days. After a time, the intervals between attacks become shorter and shorter. Occasionally they occur at certain periods of the year (spring or fall).

In the course of the disease the nerve is tender over certain definite points (*pain points*), which usually correspond to bony foramina. These points are, of the first or supra-orbital division, the supra-orbital foramen, palpebral point in the upper eyelid, the nasal point at the bony side of the nose; of the second division, the infra-orbital foramen, a point in the upper lip lateral to and below the wing of the nose, a point at the anterior portion of the temple near the malar bone; and of the third division, the mental foramen, a point over the inferior dental foramen and the zone located immediately in front of the tragus. In neuralgia of the first and third division, a point of

tenderness is found over the parietal eminence. As this borders on the domain supplied by the occipital nerve, the pain point must be ascribed to the proper nerve. Neuralgia of the trigeminus may involve only two or all of the branches of the fifth nerve. In some instances, a peripheral causative factor may be restricted to the domain of a single division (alveolar abscess), yet the pain may *radiate* to the other branches. *Irradiation of pain* of this sort may extend to distant nerve areas, such as from the auriculotemporal to the shoulder. However, radiating pain is never as severe as it is in the nerve trunk primarily involved.

Almost all of the attacks or exacerbations are attended with so-called *irritation symptoms,* which consist in redness of the conjunctiva, lacrimation, increased flow of nasal secretion and saliva, redness of the skin, sweating, and a feeling of heat. Herpes usually appears upon the forehead. Often, involvement of the facial nerve is evidenced by fibrillary contraction and spasm of the muscles of the face, and irritation of the motor root of the fifth nerve is attended with spasm of the muscles of mastication. In severe cases, a condition of general muscular spasm develops and this is associated with nausea and vomiting. Occasionally the pulse is slow.

When trifacial neuralgia heals spontaneously, the pain disappears very gradually. Unfortunately, this outcome is rare. Temporary disappearance of the symptoms is not uncommon, but usually, even after removal of the seemingly causative peripheral division, the attacks return. Krause[1] says "Of my 73 cases of peripheral resection of a division of the fifth nerve, only one seventh remained free from relapses; relief rarely lasted more than two years."

The *prognosis of trigeminal neuralgia,* strange to say, does not usually involve danger of a fatal outcome. As a rule the afflicted is well nourished, except in cases in which partaking of food precipitates an attack of pain. The outcome is largely dependent upon the cause of the neuralgia. If this is removable, recovery often follows. Operative measures of relief render the outlook better. They must not be delayed too long.

The *general causation of trifacial neuralgia* includes infectious diseases, malaria, influenza, typhus, typhoid and smallpox; systemic poisoning with mercury, lead, alcohol and nicotin; diabetes mellitus, gout and anemia. Of the chronic infectious diseases, lues must always be taken into account. No doubt luetic narrowing of the foramina of exit is often responsible for the disease.

DETERMINATION AS TO PERIPHERAL OR CENTRAL SEAT OF THE CAUSE OF THE NEURALGIA IS VERY IMPORTANT. In the event of a peripheral lesion, such as scars, foreign bodies, tumors, etc., which, according to clinical experience, is capable of producing neuralgia, one is justified in regarding this as a competent cause and in acting accordingly. If the affliction immediately follows exposure to cold or an injury of a definite area of the face and the symptoms are restricted to this zone, a causative association may be assumed. In each instance it must be remembered that an initial nerve lesion progresses toward the center. It does not require the exercise of imagination to realize that the larger the number of branches of the nerve involved, the higher up (more centrally) one must look for the causative lesion. In this connection, irradiation symptoms must be excluded. When the three branches of the nerve are involved, it is justifiable to assume that the lesion is intracranial, such as periosteal and osteal changes in the middle fossa of the skull. In instances in which neuralgia is limited to a single branch (as is usual at first), this does not by any means indicate that the lesion is peripheral (unless the latter is demonstrable), and it may simply herald the influence of a beginning central cause. The motor root of the nerve is the last to give evidence of impairment. In not a few instances, pressure on the sensory fibers of the fifth nerve is attended, not with neuralgia, but with anesthesia. Strange to say, an association between cerebral lesions and neuralgia is very rare. However, occasionally, when every effort at relief has failed, autopsy shows that a brain lesion has been the cause of the pain and that this has not been attended with any other symptoms.

The *general treatment of trifacial neuralgia* must encompass the removal of all possible causes, of which the following deserve especial attention: pathological teeth, ears and eyes, the cavities of the face, and sclerosis of the alveolar processes. Foreign bodies located near, or within, a nerve trunk must be sought for. The *medicinal treatment* should not be omitted. Of the host of remedies used, quinine and strychnia have been of actual service. Morphin and cocain are only palliative; their protracted employment always leads to an unfortunate condition of affairs. *Electrotherapy* has relieved the pain.

The *injection of alcohol* (80 per cent) into the foramen rotundum (second branch) and the foramen ovale (third branch), first used by Schlösser[2] and extensively practiced by Harris[3] and Offerhaus,[4] affords relief for varying periods of time. The success of the technic is entirely dependent upon the operator's knowledge of anatomy.

Peripheral operations are likely to accomplish the purpose if executed before the ascending changes involve additional branches of the nerve. Simple division of the affected nerve (*neurotomy*) is no longer practiced and has been replaced by extirpation of as large a segment of the nerve trunk as is possible (*neurectomy*) or, as the present procedure is called, *nerve extraction* (Thiersch[5]). The nerve is exposed and, without being sectioned, is grasped (transversely) with an especially constructed forceps, by means of which it is slowly twisted on its axis. In this way the peripheral branches and the central trunk are extirpated to an astonishing extent; in some instances a portion of the gasserian ganglion has been avulsed.

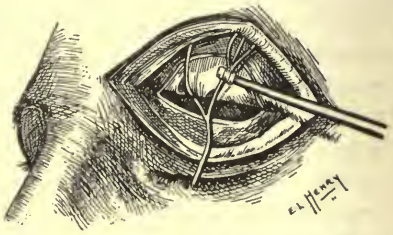

FIG. 787.— TRUNK OF SUPRAORBITAL NERVE TWISTED OUT; PERIPHERAL PORTION OF NERVE "COMING AWAY."

The *first trigeminal branch* (ophthalmic) is exposed by a curved incision 3.5 cm. in length, carried along the superior edge of the orbit, exposing the supra-orbital nerve as it emerges from its foramen or lies in the incisura (Fig. 787).

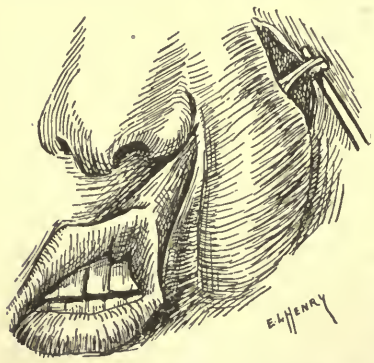

FIG. 788.— TWISTING OUT THE PERIPHERAL BRANCHES OF THE INFRA-ORBITAL NERVE.

The second *trigeminal branch* (maxillary nerve) is attacked as follows:

The incision begins ½ cm. below the inner edge of the lower margin of the orbit, runs obliquely outward and downward to the posterior lower edge of the malar bone, and is about 4 cm. in length. The upper branches of the infra-orbital nerve soon come into view at the superior edge of the wound. These are left undisturbed in their connection with the trunk, while the peripheral branches are pulled out with forceps. The infra-orbital foramen is now exposed 1 cm. below the inferior edge of the orbit, a little internal to its middle, and the periosteum is pulled down until the branches of the infra-orbital nerve are clearly visible. Next, the periosteum and floor of the orbit

are stripped backward as far as possible. Then, with a broad retractor, the orbital contents and periosteum are carefully lifted, exposing the nerve — a white stripe visible through the translucent upper wall of the canal farther back, lying free in the sulcus. The canal is opened by removing a wedge shaped section of bone with a narrow, straight chisel. The entire contents of the canal is lifted out of the bony channel on a blunt hook, as far back as the orbital fissure. However, the artery need not be isolated from the nerve unless this is readily accomplished. The forceps are introduced as far back as possible into the orbit and the nerve drawn out with a slow, constant pull. The peripheral nerves are rolled up on the clamp by a very slow, twisting motion until the finest terminal branches are delivered from the cheek, the upper lip, and the ala of the nose (Fig. 788).

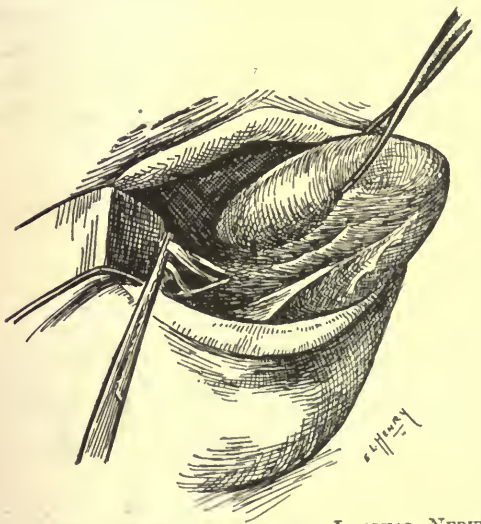

FIG. 789.— EXTRACTION OF THE LINGUAL NERVE.

The branches of the third, or *inferior maxillary, division* may be removed separately.

The *lingual* nerve is extirpated by exposing it as it enters the base of the tongue, i. e., between the ramus of the lower jaw and the *palato-glottidean fold.* It lies in the neighborhood of the last three molar teeth, just under the mucous membrane, exactly at the point of transition from the side of the tongue to the floor of the mouth. With the mouth held widely open and the tongue drawn forward and upward, the mucous membrane is incised at the point stated (Fig. 789). The nerve is immediately visible and is grasped with forceps and twisted out.

The *auriculotemporal nerve* is easily reached through a small vertical incision at the upper angle of the parotid gland. The nerve is accompanied by the superficial temporal artery, from which it is readily separated; it is then grasped with forceps and twisted out.

The *superior maxillary nerve* may be exposed at its exit through the

foramen rotundum only by osteoplastic resection of the zygoma and dissection into the sphenomaxillary fossa. The skin incision begins a finger's breadth outside and below the end of the eyebrow, descends along the posterior edge of the frontal process of the malar bone and thence, in a flat arc (concavity upward), backward and downward to the lower edge of the zygoma and along this to the anterior edge of the articular tubercle. Here the incision turns obliquely upward, ending just above the zygoma in front of the ear. The zygoma is now exposed along its medial surface with the elevator, and, close to the articular tubercle, is divided with bone forceps. When the skin is retracted downward, the branches of the facial nerve are placed out of danger and the zygoma may be divided. The anterior origin of the zygoma is now sectioned and the bone thus liberated is turned down (Fig. 790). The approach to the sphenomaxillary fossa is now free; only the anterior fibers of the temporal muscle need be divided. Proceeding into the depths with small sponges and a blunt elevator, the surgeon advances along the posterior surface of the superior maxilla to reach the sphenomaxillary fossa. The fat and the plexus of veins are pushed aside, and the internal maxillary artery is carefully freed and pulled backward with a retractor. The inferior orbital fissure is located with a probe and any fat which interferes with the view is carefully removed. The nerve now comes into view where it emerges from the foramen rotundum and runs obliquely forward and outward through the inferior orbital fissure; here it is hooked up (Fig. 790) with a small tenaculum, separated by blunt dissection, and lifted away from the infra-orbital artery. It is now grasped with the forceps and cut close to the foramen. The wound is drained and the zygoma sutured back into place (Lücke,[6] Braun,[7] v. Friedländer[8]).

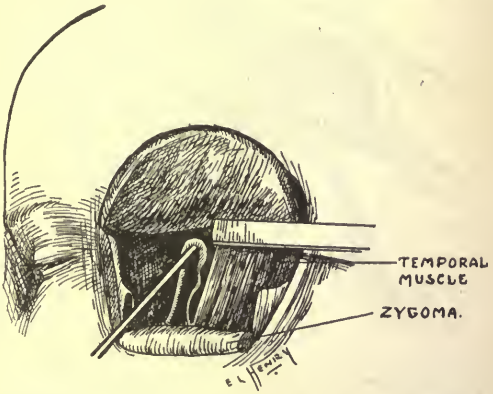

FIG. 790.— EXTRACTION OF THE SUPERIOR MAXILLARY NERVE.

The zygoma is turned down and the temporal muscle retracted. The nerve is lifted with a blunt hook.

The *third branch of the trigeminus* may be reached at the *foramen ovale* as follows:

The incision of approach is very similar to that employed in attacking the second division. The zygoma, together with the parotido-masseteric fascia and masseter muscles, is turned down, exposing the coronoid process of the lower jaw. This is freed from muscle with an elevator and cut off at its base with bone cutting forceps, exactly on a line with the deepest portion of the sigmoid notch. The loosened process, together with the attached temporal tendon, is turned up, exposing the infratemporal fossa and at the same time the external pterygoid muscle and the internal maxillary artery, which runs between the two points of origin of the muscle. The pterygoid muscle is pulled upward and divided and the internal maxillary artery is cut between two ligatures. The external plate of the pterygoid process is cleared with a raspatory to the point where its root joins the base of the skull at the foramen ovale. The large nerve trunk is seen coming out of the foramen; it is seized with the nerve forceps (Fig. 791) and torn out of the cranium. The otic ganglion may be removed at the same time. The wound is drained and the zygoma replaced and stitched into place (Kocher,[9] Krönlein,[10] and Krause[11]).

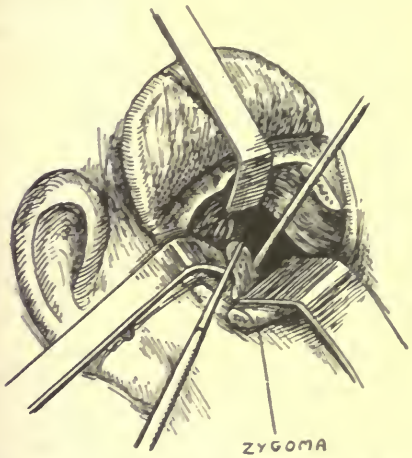

FIG. 791.—THE INFERIOR MAXILLARY NERVE LIFTED ON A DIRECTOR AND SEIZED WITH A NERVE CLAMP JUST BELOW THE FORAMEN OVALE.

Intracranial removal of the ganglion of Gasser is a measure of considerable magnitude. Its indications are taken up at the beginning of the chapter. Of the various methods, that of Krause[11] will be described here. For additional methods the reader is referred to the bibliography (Rose,[12] Dollinger,[13] Cushing,[14] Lexer[15]). The technic is divided into three acts.

The *first act* consists in *opening the cranial cavity.* The *incision in the scalp* begins immediately above the zygoma close in front of the tragus, runs convexly backward and upward, forms a half circle, and

curves downward, back to the anterior aspect of the zygoma. The flap thus outlined measures at its base 3.5 cm., is 6 cm. in height and is about 5 cm. at its widest portion (Fig. 792). The entire operation is extradural. The periosteum is slightly pushed back from the bone; a single hole is made in the cranium with a Doyen burr (Fig. 338), and the bone sectioned in the line of the scalp incision with the Dahlgren craniotome (Fig. 753) or a de Martel osteotome (Fig. 755). The base of the osteoplastic flap thus formed is forcibly broken with any appropriate lever, exposing the dura. The line of fracture is usually located about 1 cm. above the zygoma. The lower edge of this is removed down to the base of the skull with rongeur forceps. This is imperative. The flap now hangs down upon the face and is out of the way. Krause[11] regards temporary resection of the zygoma as unnecessary.

The *second act — ligature of the middle meningeal artery*—is now undertaken. The finger or a raspatorium separates the dura from the base of the skull. If a protruding eminentia capitata is in the way, it is chiseled off. The foramen spinosum and the artery traversing it soon come into view. The brain is held upward by an assistant with a broad spatula and the vessel, carefully isolated from its embedding dural processes, is grasped with two narrow bladed artery forceps and cut between them and tied. If this does not succeed, the foramen may be plugged with gauze and the operation may proceed.

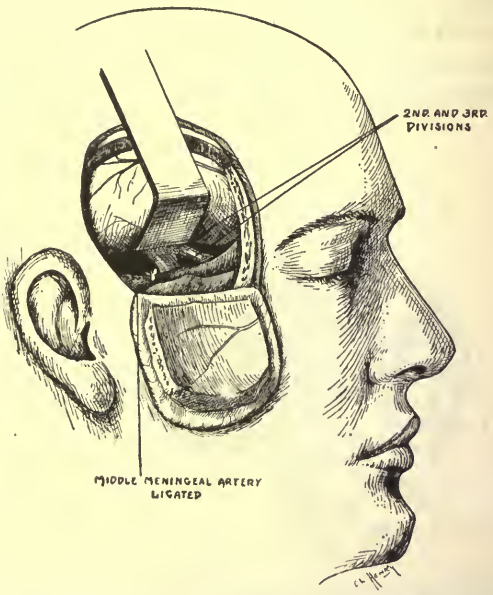

FIG. 792.— EXTIRPATION OF THE GASSERIAN GANGLION.

The second and third divisions exposed. The middle meningeal artery is tied.

The *third act — exposure of the ganglion and its removal* — is accomplished as follows: The dura is progressively elevated (inward) by means of gauze pledgets held in artery forceps, until the third, and

then, considerably farther on, the second, division is exposed from the ganglion to the foramen rotundum and ovale. A little more manipulation, supplemented perhaps by snipping of dural adhesions, brings into view the entire ganglion. If difficulty is experienced, the third branch is grasped with forceps and pulled outward, thus making the ganglion more accessible. The first branch, because of its close relationship with the cavernous sinus, is exposed only as a part of the ganglion. The ganglion is now grasped at its posterior (central) aspect with the nerve forceps and the second and third divisions carefully divided at their respective foramina of exit. By slowly rotating the forceps, the ganglion, together with a goodly section of the first division, is readily "torn out." The stumps of the second and third branches are pushed into the foramina. The brain is allowed to fall into place, a drain is inserted and the flap sutured as usual. The drain is removed on the fourth day. The trophic disturbances which occasionally occur in the eyeball are obviated by keeping the eyeball covered during convalescence. Of 85 cases operated upon by Krause,[1] ten died as the result of the operation.

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CHAPTER V

SURGERY OF THE SALIVARY GLANDS

Injuries of the Salivary Glands.— Of the injuries of the salivary glands, only those of the parotid are of special importance. Trauma to the others, aside from complicating infection, even though followed by the formation of a fistula, causes no disturbances, as the vicarious opening always communicates with the mouth.

Severe injuries of the parotid (stab and gunshot wounds) are likely to be attended with involvement of the facial nerves and trauma to important vessels. The latter is associated with much bleeding. In cases of this sort, an external salivary fistula often forms at the end of a few days. However, most of these heal spontaneously. *Primary union* in the wound is the best *prophylactic measure* as regards the occurrence of a fistula. During repair, mastication must be avoided; the patient subsists on fluid diet.

Injuries of the parotid duct are more troublesome. As a rule the duct is divided by a cutting instrument, but it may be contused and subsequently may slough. The *diagnosis* is made by introducing an irritating substance into the mouth and noting the discharge of saliva from the wound.

The *treatment*, in *non-penetrating wounds* in which the duct is divided, consists in its careful repair by suture. In *penetrating wounds* only the superficial wound is closed; the duct then establishes a fistulous opening within the mouth.

Salivary fistulae (parotid fistulae) after injury or pathological destruction of the parotid gland substance are common. Here again, however, spontaneous healing is not rare. The fistulous opening may present at any portion of the surface of the gland. The orifice of a fistula of this sort is usually surrounded by an area of eczema due to the irritation of the discharge.

The *treatment* is primarily directed toward causing inflammatory obliteration of the opening. This may often be accomplished by the application to the fistulous tract every four or five days of the *silver*

stick, or, better still, by a single application of the actual cautery. If these measures fail, the tract is excised and the wound closed with a plastic flap. In obstinate cases a portion of the gland may be extirpated.

Salivary duct fistulae (fistulae of the parotid duct) cause much more disturbance than those of the gland and rarely heal without operative help. A fistula of this sort may be regarded as permanent (1) when the *buccal mucosa is adherent to the skin;* (2) when the *peripheral end of the duct is obliterated;* (3) when *large defects are present in the duct.*

The *treatment* of permanent fistulae depends upon their seat, i. e., *buccal, masseteric, or glandular.* In 1895 Delarue[1] tabulated twenty-six methods of treating the affliction. Relief may be afforded by (1) *reëstablishment of the normal path of secretion;* (2) *converting an external fistula into an internal one;* (3) *destroying the secretory function of the gland.*

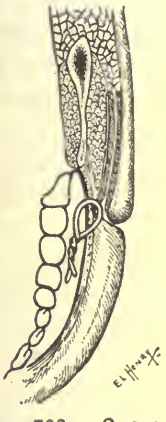

FIG. 793.—OPERATION FOR SALIVARY DUCT FISTULA (Deguise).

Reëstablishment of the normal path of the secretion is a delicate operation. It means dissection of the duct and its repair by suture (Nicoladoni[2]). *Converting the outer fistula into an internal* one is the method of choice. It is desirable to implant the peripheral ends of the central portion of the duct into a slit in the mucosa. As this is not often feasible, the *single* or *double perforation* of the mucosa is employed.

Single puncture (Desault[3]) consists in perforating the mucosa at the site of the fistula with a trocar or or with the galvanocautery. The canal thus formed is kept patent by the introduction of a drainage tube until the internal flow of saliva is assured (8 to 10 days).

Double puncture of the buccal mucosa is very simple (Deguise[4]) and is much practiced. One half of the diameter of the buccal side of the fistula is transfixed ("in and out," ½ cm. apart) by a cannula through which a silver wire is introduced. The latter is twisted (Fig. 793) and gradually cuts its way out. The outer wound usually heals spontaneously, though there is no objection to refreshing and suturing it.

Direct implantation of the central end of the duct into the mucosa of the mouth is accomplished by dissecting out its central portion and

sewing it into a stab wound in the buccal mucosa. The method succeeds when enough of the duct is left to carry out the technic.

The *abolition of the secretion of the gland* is based on experimental work (Langemak[5]), showing that ligature of the duct causes the gland to cease functioning. In man the measure is followed by a grave reaction in the gland, including sloughing and infection (see also Rousseau[6]).

Foreign Bodies and Calculi.— *Foreign bodies* gain access to the ducts of the salivary glands from the mouth, or from without. Of the ducts, that of the *submaxillary gland* is most often visited by a foreign body, usually a fish bone, a hair, a bristle, a feather, etc.

The accident is attended with rather *stormy symptoms,* especially when the unwelcome visitor becomes coated with lime salts. The *gland swells* (*intermittently*) and is exceedingly painful. After a time the clinical picture of *inflammation* develops and this often ends in *abscess* and subsequent *fistula* formation.

The *diagnosis* is certain only when the foreign body is palpable. The value of the Röntgenogram depends upon the character of the obstructing agent.

The *treatment* should of course be directed toward removal of the offending substance. This may be accomplished by incising the duct. In some cases opening of an abscess liberates the body. If a submaxillary fistula persists, the gland may be extirpated *in toto* — not a difficult procedure.

Salivary stone (sialolithiasis) is rare; it attacks the ducts more often than the glands. The calculus consists of an organic nucleus and an inorganic deposit arranged in layers. Del Fabbro[7] and Kroiss[8] have shown that the calculi are developed as the result of inflammatory processes in the glands (not the reverse, as has been believed). The pathological process alters the character of the salivary secretion, and the inorganic salts are deposited upon a bacterial nucleus (Klebs,[9] Galippe[10]).

The *clinical picture* of sialolithiasis varies. The concretions may lie quiescent for years and then suddenly obstruct the duct. *Intermittent salivary tumor* is characteristic. The swelling is very painful (*coliques salivaires* of the French) and usually develops during mastication. Occasionally, a sudden discharge of saliva and pus into the mouth heralds the end of an attack. Not a few cases develop an acute inflammatory process in the gland and this often terminates in abscess formation. When the stone (or stones) is located in the gland, the

latter picture develops primarily. In this event, the process must be diagnosticated from alveolar periostitis, not always an easy task.

In the *treatment* an effort should be made to *remove the stone through the mouth.* The exceptions to this rule are when a fistula leads to the concretion; when the stone lies in an external abscess; and when the stone is intraglandular. Removal through an incision in the mouth is readily accomplished. When, however, the concretion is *deeply located in the sublingual* gland, removal may be difficult. A subsequent intrabuccal fistula is unimportant. *Stones located in the substance of the submaxillary or parotid gland* cannot be removed through the mouth, but must be enucleated from without by careful dissection. Total extirpation of the submaxillary gland is not objectionable. However, this is not justifiable as far as the parotid is concerned.

Acute Inflammatory Processes of the Salivary Glands.— Acute inflammatory processes of the salivary glands are divided into *primary and secondary.*

Of the *acute primary inflammations, mumps* (*parotiditis epidemica*) is the most frequent. Its discussion belongs to medicine. However, the occasional *development of abscess* in the gland, which demands incision, justifies brief allusion to it. More important perhaps is the not uncommon complicating *orchitis* which also at times goes on to *abscess formation* and demands drainage; or, if sloughing occurs, the testicle may have to be enucleated.

Acute secondary inflammation of the salivary glands is more likely to be of surgical interest. It occurs in connection with *local or constitutional processes.* Of the local causes, *foreign bodies and stones* (see above) and *extension from contiguous infectious processes* (suppurative lymphadenitis) and various forms of *stomatitis* (especially mercurial) and *trauma* are the most important.

Parotitis frequently occurs as a part of a *general infectious disease,* such as typhoid fever, scarlatina, pneumonitis, influenza, variola, etc. It is most frequently seen in association with typhoid fever. Powers[11] reports a case due to the gonococcus.

The *cause of secondary inflammation of the salivary glands* is ascribed to infections of the mouth which lessen the flow of saliva and thus favor invasion of the gland tissue. Pawlow[12] has shown that the flow of saliva is diminished after all abdominal operations. That narcosis is not responsible for the process is shown by the fact that it develops after operations performed under spinal anesthesia. The

notion that parotitis is most frequently a part of a bacteriemia has recently gained ground (see v. Mikulicz and Kümmel[13]). It is certain that postoperative parotitis does not often occur when the mouth is thoroughly cleansed before the operation (Haubold[14]).

In describing the *clinical picture* only the inflammatory processes occurring in connection with infectious diseases and after operations are taken into account; the other forms are taken up with foreign bodies and calculi. The *beginning* of the disease closely resembles an acute infectious disease. It usually develops early in the course of a systemic infection and as a postoperative complication makes its appearance from the third to the fifth day, rarely as late as the tenth. When the *parotid* is affected, the gland swells, is very painful and becomes tense. The swelling often extends from the temporal region to the neck. The appearance of the patient (especially when both parotids are involved) is very characteristic. The skin is red, edematous and immovable. Deglutition, mastication and respiration are painful. The symptoms continue to increase for three or four days, when either resolution occurs or an *abscess develops.* The density of the embedding connective tissue does not permit of diagnostic fluctuation. Abscess formation is likely to be multiple and is often attended with necrosis of considerable areas of gland tissue. Extension of the process in the form of *pyemia* or proliferation into the visceral cavities, including the cranial cavity, is not rarely responsible for a fatal outcome. The connection of the posterior aspect of the parotid with the esophageal and retropharyngeal spaces and the mediastinum favors extension of the process. This is also true with regard to the jugular vein, the lateral sinus, and the middle ear.

Infection of the *submaxillary* and *sublingual* glands is rare. In neither instance does the picture vary from that described in connection with the parotid gland, except perhaps with regard to the intensity of the process and its complications.

The *treatment* is primarily prophylactic. In all infectious diseases and as a part of preparatory and postoperative care, the mouth is carefully cleansed. When the inflammation of the salivary gland is established, Bier's hyperemia (p. 249) may be employed. This is advantageously supplemented by the application of iodin. Progressive inflammation justifies immediate unburdening of the gland with the knife (see treatment of inflammation, p. 241). In attacking the parotid gland, only the skin and the fascia are divided; the parenchyma of the gland is invaded with blunt forceps. The incision must

avoid the facial nerve. Counteropenings should be made. The *submaxillary* and *sublingual* glands may be widely opened.

Chronic inflammatory processes most often develop as a sequel to acute forms of the disease; ultimately suppuration occurs and this is drained as stated above. Recurrent attacks justify removal of the submaxillary and sublingual glands. In the parotid, resection of portions of the gland is as far as the surgeon is justified in going.

Actinomycosis — Tuberculosis — Lues.— *Antinomycosis* of the salivary glands was described by Müller[15] in 1903. It is usually secondary to actinomycotic infection of the soft parts and the bones of the face. Clinically, it appears in the form of a chronic inflammatory process.

Tuberculosis of the salivary glands occurs despite the long existing belief that these organs are exempt from the disease. Küttner[16] has collected twenty-seven cases. Wood[17] thinks that the infection enters through the tonsil. See also Legueu et Marieu,[18] Parent,[19] de Paoli,[20] Homuth.[21]

The process is attended with circumscribed enlargement of the affected gland. These areas are very likely to break down and form fistulae. A *clinical diagnosis* is impracticable and reliance must be placed upon the microscope.

The *treatment* consists in operative removal of the process.

Lues of the salivary glands is not as rare as tuberculosis; most of the lesions in twenty cases collected by Küttner[16] were in the parotid.

The *clinical picture* is one of chronic inflammation with hyperplasia of connective tissue. When this is present, together with other luetic manifestations, diagnostic therapy is justified. See also Fournier,[22] Claus.[23]

Salivary Cysts.— Retention of salivary secretion occurs in connection with inflammatory processes, the presence of foreign bodies, and calculi in the ducts, or as the result of obstruction of the latter by scar tissue, etc.

Cystic enlargement of the ducts occurs in the submaxillary and parotid glands. The condition is ascribed to *acquired* cicatricial or *congenital* obstruction. The affected duct is usually symmetrically dilated and as a rule the gland itself is more or less enlarged. The superimposed skin and mucosa is quite normal in appearance. Occasionally, a cystic enlargement of this sort is lined with a layer of lime salts.

Pressure on a cyst of this sort, when the duct is not entirely occluded, causes it to empty into the mouth. When the duct is closed, irritation of the mucosa provokes a flow of saliva only from the opposite side. Strange to say, the dilated duct does not reach great size; probably the gland ultimately ceases to function. In other cases, an abscess forms and a *salivary fistula* develops. When an opening occurs in the mucosa it makes no difference; an opening in the skin, however, is followed by the clinical picture described above.

The *treatment* aims at the establishment of a vicarious opening into the mouth, which is best accomplished by the Deguise method (p. 1629).

Cysts of the salivary glands are due to obstruction of one or more of the subdivisions of the main secretory duct and are classed with the retention cysts. The obstruction is usually due to an inflammatory process. Cysts of this sort are not common. No doubt so-called ranula belongs to this class.

FIG. 794.— RETENTION CYST OF SUBMAXILLARY GLAND.

Retention cysts of the parotid and submaxillary glands (for sublingual see ranula) are small (size of a hen's egg) and usually solitary, although multilocular cysts of the parotid are reported (Kroiss[8]). Cysts of this sort grow slowly. The *diagnosis* is made with the *aspirator*. The *treatment* consists in extirpation of the cyst.

Echinococcus cysts of the salivary fistula are very rare (v. Mikulicz and Kümmel[13]).

Tumors of the Salivary Glands.— New growths of the salivary glands possess considerable surgical importance. It is not impossible that most of these are of endothelial origin. On the other hand, all of the

tumors discussed in Part VI are found in these organs and present a clinical picture, the character of which is especially influenced by the location of the lesion.

Hemangiomata grow very rapidly and demand early and prompt excision. Injections of alcohol, etc., never accomplish the purpose. *Lymphangiomata* are rare (Lannelongue et Achard[24]).

Lipomata, fibromata, myxomata and sarcomata do not differ from those of the other organs.

Mixed tumors are the most frequent growths found in the salivary glands; the parotid is most often affected. A considerable number of tumors of this sort has involved the accessory parotid.

FIG. 795.—MEDULLARY CARCINOMA OF PAROTID GLAND.

Clinically, mixed tumors of the salivary glands appear primarily as a circumscribed growth, but soon involve the entire gland structure. Their motility under the skin is characteristic. The disturbances tumors of this sort produce relate to pressure and interference with mastication, speech, deglutition, and respiration. In a certain number of cases the tumor becomes malignant in character (Wilms[25]).

The *treatment*, as one can never be certain of the exact character of the growth, consists in removal of the entire gland. When the tumor is located in the parotid, this may be modified with the view of saving the facial nerve.

Carcinomata occur less frequently than mixed tumors. Both the scirrhous and true medullary carcinomata develop in this situation. The *scirrhous form* grows slowly and runs a clinical course similar to that of like growths of the mammary gland. The facial nerve is involved early. The overlying skin is soon involved and in many instances presents a condition of infiltration which the French call "squirrhe en plaque" (Michaux[26]). The lymphatic glands are involved late. The *true medullary form* is richly cellular, grows rapidly (Fig. 795), and soon perforates the skin. This is followed by the

events discussed in connection with carcinomata in general (Part VI, chap. xvii).

Malignant tumors of the salivary glands must be diagnosticated from inflammatory processes, especially granulomata, tuberculosis, and lues. A very malignant form of sarcoma, which rapidly invades the skin, presents a clinical picture not unlike that of carcinoma.

The *treatment* consists in total extirpation of the affected gland.

Total Extirpation of the Parotid and the Submaxillary Glands.—Extirpation of the parotid is a difficult operation. In malignant disease *extracapsular* enucleation is employed; the *intracapsular* method is permissible for the removal of benign processes. In the former class of cases the *facial nerve* must be sacrificed; in the latter it may be preserved.

The *skin is sectioned* vertically close to the tragus, from the level of the external ear to 4 or 5 cm. below the angle of the jaw; this is supplemented by a horizontal incision a finger's breadth below the zygoma, which extends forward about two and one half inches. The flaps are dissected back and the surface of the gland is exposed. Extirpation of the gland is begun at its lower pole, where the lymphatic glands are found and removed; here the large veins are tied and the gland tissue is lifted from the external carotid artery. The latter is divided between two ligatures. The gland is next separated at its anterior border from the masseter muscle. Removal of the posterior portion of the gland is not easy; it is slowly and carefully accomplished, each divided vessel being at once ligated. By careful dissection the gland is separated from its surroundings until it is held by its attachment only to the pharynx and styloid process, from which it is finally dissociated with great care. The *vessels divided during the operation are* the external jugular, the anterior and posterior facial veins, the transverse facial, the posterior auricular, the occipital, the superficial temporal, the anterior articular, the zygomatico-orbital, the internal maxillary, and the external carotid arteries. The *facial nerve* is sacrificed.

Enucleation of the submaxillary gland is not difficult. A curved incision (concavity upward) is made immediately below the lower edge of the jaw (from the symphysis to the angle). The platysma is divided, the gland is lifted from its bed, the facial artery and vein are divided between ligatures, the attachments of the gland to the jaw are cut, and the liberated organ is delivered. The labial branch of the facial nerve is usually cut. Care must be taken not to injure the contents of the

digastric triangle. The deep cervical glands should be removed when the operation is performed for malignant disease.

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CHAPTER VI

DISEASES OF THE TEETH AND OF THE GUMS

While the affections of the teeth and gums have become specialized under the head of dentistry, there are certain aspects of the problem with which the surgeon should be familiar.

Caries of the Teeth.— Caries of the teeth is the most widely distributed disease of civilized man. Its underlying causation is undoubtedly attributable to the refinements of the art of cookery. The mastication of firm substances, first on one side of the mouth, and then on the other, is the most certain prophylactic measure in this connection. Incidentally, this also reduces the bacterial flora of the mouth.

The additional *predisposing causative factors* are as follows:

Abnormally close proximity of the teeth, favoring retention of foreign substances; *pregnancy* and *lactation; systemic diseases,* such as diabetes, chlorosis, and the general infections, and *heredity.*

The *exciting cause of caries* consists in bacterial invasion of the tooth and its simultaneous softening. The latter is the outcome of the fermentation of retained carbohydrates produced by the bacteria.

When the carious process has destroyed the cortex of the tooth, exposing the dentine, the disease progresses rapidly and cavities are formed which ultimately extend to the tooth pulp.

Caries may be prevented by mechanical cleansing of the teeth, the avoidance of easily decomposed food, and, most of all, the proper physiological use of the teeth in mastication.

Pulpitis occurs when a carious cavity extends into the medulla of the tooth. As the tooth pulp is richly supplied with nerve endings, an inflammatory process in this situation is exceedingly painful. The *treatment* consists in cleansing the cavity, removing the detritus and pulp (if necessary), and filling the carious cavity. Chronic pulpitis often demands extraction of the affected tooth.

Periodontitis and alveolar periostitis is the next step after pulpitis, the infection extending to the tooth socket. In accord with the port of infection, periodontitis is divided into *marginal* or *apical,* both of

which may be *acute* or *chronic*. The *treatment* consists in drainage of the infected area. If the process is extensive, the offending tooth must be extracted. The general belief that a tooth should not be extracted during an acute process is a relic of the dark ages.

The *extraction of a tooth* is a minor operation that the surgeon is frequently called upon to perform. It should be remembered, however, that the unnecessary sacrifice of a tooth should be avoided, and that the need for it must be great before extraction is justified. The technic of the measure is divided into five acts: (1) cleansing the surroundings of the doomed tooth; (2) application of the forceps; (3) luxation movements; (4) extraction; (5) after treatment of the wound. Cleansing is best accomplished with alcohol. In applying the forceps care must be taken not to grasp the gum nor include the alveolar process. The luxation movements consist in lateral displacement of the tooth which is thus loosened and is readily *lifted, not twisted*, out of its socket. The after treatment consists in cleaning the cavity with 3 per cent carbolic acid solution, which is followed by the application of iodoform powder. If bleeding is excessive, the cavity is tamponed with iodoform gauze saturated with adrenalin solution (1:1000) or with thromboplastin (see Hemophilia, p. 1063).

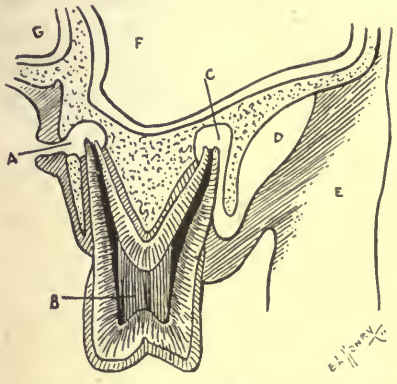

FIG. 796.— DIAGRAM OF ALVEOLAR ABSCESS, RESULTING FROM DISEASE OF MOLAR TOOTH (after the American System of Dentistry).

A, Abscess arising from escape of septic material from *B*, the pulp chamber, through the foramen at apex of the fang; it has burrowed directly through the alveolar process and burst through the gum; *C*, similar abscess, which has tracked down between the tooth and the alveolus, and spread out beneath the alveolar periosteum at D, constituting the typical alveolar abscess; *E*, cheek; *F*, antrum; *G*, nasal cavity.

Diseases of the Gums.— Diseases of the gums, other than the processes occurring in connection with lesions of the mouth, are due to extension of infection from the teeth (caries). The most common of these, and the one properly alluded to here, is known as *alveolar pyorrhea*.

Alveolar pyorrhea simultaneously attacks the alveolar processes, the covering of the roots of the teeth, and the gums. It is attributed to a lessened resistance to bacterial invasion on the part of the tissues

involved. The disease is characterized by a loosening of the gums from the teeth and the alveolar processes. As a rule, the neck of the tooth is surrounded by a ring of lime deposit (tartar). Pressure on the gum expels a few drops of pus. The root of the tooth is usually covered by a layer of granulation tissue, the outcome of a low grade of infection. After a time, the alveolar processes are absorbed or destroyed, the teeth are loosened and ultimately fall out.

The *treatment* of alveolar pyorrhea is directed toward combating the local infection and improving the patient's general condition. The former is essentially the work of the dentist. However, it is not out of place to state here that there is NOTHING MORE EFFECTIVE IN THIS CONNECTION THAN FREE EXPOSURE OF THE INFECTED AREAS AND THE LIBERAL USE OF THE THERMOCAUTERY.

CHAPTER VII

TUMORS OF THE JAWS

It is of practical value to divide tumors of the jaws into those etiologically connected with the development of the teeth and those independent of this origin. Those of the latter class are similar to those appearing in other portions of the body.

Odontogenous Maxillary Tumors.— Follicular dental cysts appear in early life. They develop in the interior of the maxillae and, as they slowly enlarge, destroy the embedding bone until the latter is only a thin shell. These cysts possess a smooth wall lined with fibrous tissue covered with a layer of epithelium, and contain a clear fluid. The lesion is believed to represent the cystic degeneration of a tooth bud. The tumor is usually covered with a perfectly smooth mucosa, which is more or less elevated in a globular form. The *diagnosis* is possible only with the help of the Röntgenogram or the aspirator. *Excision of the anterior wall of the cyst* is followed by its spontaneous obliteration.

ADAMANTINOMATA (multilocular cystomata and benign central epitheliomata of the maxillae).— *Multilocular cystomata,* like maxillary cysts, originate in tooth buds. The cysts are found distributed throughout the entire bone and usually communicate with each other; the superimposed skin and mucosa is normal in appearance. On the other hand, the bone is gradually converted into a thin shell, and, in places, disappears entirely, so that the cyst wall lies in contact with the mucosa and the skin. Their structure is very like that of a follicular cyst.

The *solid adamantinomata* are encapsulated within a thin bony wall. The histology of these tumors is taken up in Part VI.

Clinically, both the cystic and solid adamantinomata present the picture of benign growths. The *diagnosis,* in the cystic form, is based on the presence of a fluctuating tumor and the Röntgenographic findings; the solid form can be differentiated from sarcoma only by means of microscopical examination of an excised portion of the growth.

Enucleation of the growth is usually followed by return of the

process and this can be prevented with certainty only by excision of the invaded jaw (Delsaux[1]).

Odontomata.— The term "odontomata" is applied to tumors which spring from fully developed teeth. They owe their origin to changes in the dental anlage of one or more teeth. Tumors of this sort usually appear in the lower jaw; they are encapsulated and rarely grow larger than a hen's egg. They consist of cement and dentin, usually superimposed upon an unerupted tooth. According to Malassez (quoted by Galippe[2]), the process is due to changes coincident with the retention of a tooth or teeth (see also Cousins[3]). The tumor is easily removed after chiseling away the superimposed shell of bone.

Epulis: Epulis Fibromata and Epulis Sarcomata.— The term "epulis" is properly applied only to tumors of the gums of fibrous and sarcomatous nature which, despite their histological differences, present an almost uniform clinical picture.

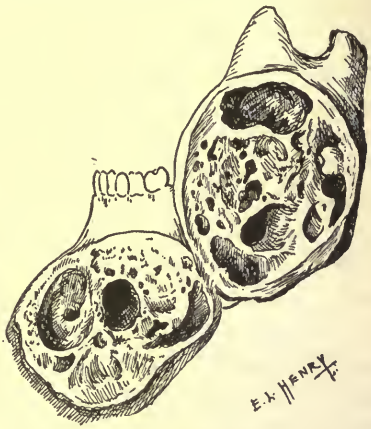

Fig. 797.— Multilocular Cystoma of Jaw.

Epulis originates from the periosteum of the alveolar processes and is attached by either a narrow or a broad base. It is located on both sides of the affected maxilla or on both maxillae (opposite one another). The tumor is the same color as the gum, or perhaps slightly darker. Its consistency varies, though most of the tumors are soft. Hardness argues for a fibromatous, and softness, for a sarcomatous, growth (especially giant cell). The teeth in the area of the growth are usually loose. As a rule tumors of this sort grow slowly; occasionally, a sarcomatous lesion extends rapidly (Speese[4]).

In most instances, epulis exhibits the characteristics of a *benign tumor;* it does not form metastases, nor does it return when removed.

Removal of the growth at its base is followed by return. The periosteum or periodontium from which the lesion originated must also be extirpated. Resection of the alveolar process is desirable.

Fibromata, osteomata, chondromata and *angiomata* do not differ from those found elsewhere in the body (see Tumors, Part VI).

Sarcoma.— Sarcoma is more likely to involve the upper than the

lower jaw and occurs in women oftener than in men. It is essentially a disease of early life. Its traumatic origin is extremely doubtful. The difference between *peripheral* and *central* sarcoma is especially marked in tumors of the lower jaw. In the former the jaw bone is embedded in the growth, in the latter, the bone embeds the tumor. The *prognosis* depends upon the histology of the process. Sarcomata of the jaws do not display a marked tendency to metastasize. Of the forms of sarcoma, the *melanotic* is the most malignant, *round cell* slightly less so, and the *giant cell* the least.

The *clinical manifestations of periosteal sarcoma* are those of an extending growth upon the surface of the bone; the tumor is not covered with bone. In *central sarcoma* the growth enlarges the bone itself, at first in the form of a circumscribed swelling, especially toward the periphery and the gums. In the beginning it is covered with a shell of bone tissue, but this is soon perforated. Before this happens, "parchment crackling" may be elicited. As the process goes on, the bone is more or less extensively destroyed and the soft parts are invaded. The advance of the lesion is attended with symptoms of pressure on the various nerves. Tumors located in the upper jaw occlude first the contiguous nostril, then encroach upon the orbit, and last protrude toward the face.

FIG. 798.— EPULIS.

The *diagnosis* is only certain when based upon the histological findings.

The *treatment* consists in resection of the affected jaw. This general rule may be varied with respect to sarcomata limited to the alveolar processes, in which event these may be removed (Fairbanks,[5] Hewson[6]).

Carcinoma.— Carcinoma of the jaws develops from the epithelium of the gums and the mucosa of the alveolar processes and the palate. It appears in the form of a *papillomatous growth* or an *ulcer* with hard edges. Histologically, carcinomata in this situation are of the flat epithelial type. The *central* type of carcinoma originates in the mucosa of the maxillary antrum. Of course, in all their aspects the growths extending from contiguous tissue (secondary) attack the jaws.

Carcinoma of the alveoli usually appears at the edge of the gum in the form of a hard, crater-like ulcer which soon invades the bone, and

the teeth fall out. In the upper jaw the process soon perforates the antrum. In the lower jaw spontaneous fracture occurs early. Pain is not an obtrusive symptom.

Central carcinoma causes severe pain at the outset. Presently the mucosa becomes edematous and soon the growth makes its appearance externally. The subsequent sequence of events is similar to that described in connection with sarcoma. As the carcinomatous infiltrate progresses, the osseous structure is destroyed until finally the inevitable picture described in the general part of this work (Part VI) closes the scene.

FIG. 799.—SARCOMA OF THE UPPER JAW (Johnson).

ONLY THE MOST RADICAL OPERATIVE MEASURES OF RELIEF ARE TO BE CONSIDERED. These consist in TOTAL RESECTION OF THE INFERIOR OR THE SUPERIOR MAXILLA. In the latter instance the floor of the orbit should be preserved (Hotchkiss,[7] Koenig,[8] Morestin,[9] Tarumianz[10]).

Hyperostoses.—Hyperostoses resembling tumors are not infrequently found involving the jaw bones, especially the upper. No doubt, some of these cases represent the initial stage of *leontiasis ossea* (p. 1072). The lesions are of surgical importance in the sense that they make pressure upon the soft parts, which seriously interferes with function (Fraenkel[11]).

Tuberculosis of the Jaws.— Tuberculosis of the jaws is either the *outcome of extension* from contiguous parts or is *primary* (hematogenous) in origin. The former usually arises in connection with tuberculosis of the alveolar processes, which has its origin in infection of

the gums. The process soon ulcerates and presents the characteristic grayish red nodules upon its excavated surface; sequestration of the alveolar processes is common. As a rule, the submaxillary lymph nodes are swollen. In a certain number of cases, tubercle bacilli may be found in the exudate. The *prognosis* depends upon the degree of coexisting pulmonary tuberculosis, with which the process is so frequently associated. In any event, the indication as to *treatment* is clear. The process is radically excised and the operative area is treated with iodoform paste. Occasionally, the infection takes its origin from a carious tooth and a subsequent *tuberculous pulpitis* (Michel[12]).

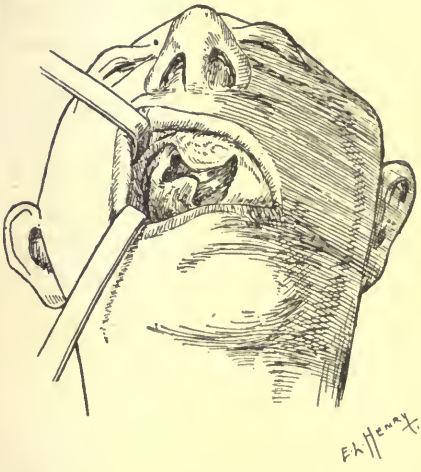

Fig. 800.— Carcinoma of the Roof of the Mouth.

A special form of tuberculosis appears as a *granulomatous process* originating in the mucosa of the maxillary antrum. In this class of cases, the lesion does not break down, but gradually distends the antrum in a manner closely resembling sarcoma. In the few reported cases, resection of the affected superior maxilla was followed by recovery (Perrier[13]).

Differentiation between carcinoma of the alveolar processes, tuberculosis, actinomycosis, and lues is not always an easy task; it requires the assistance of the microscope and the serological findings.

Actinomycosis of the Jaws.— Actinomycosis of the jaws usually begins in the mucosa of the mouth. It is divided into a *peripheral* and a *central form*. In the former the jaw is invaded by extension from the mouth. The *central form* begins as a central caries or a central new growth and is usually located in the lower jaw. No doubt, the infection gains access to the bone through carious teeth. After a time, the infection extends to the cortex of the bone, which it perforates and thence invades the surrounding soft parts.

The *diagnosis* can be made with certainty only when the microscope detects the ray fungus (p. 404). In central actinomycosis, sarcoma will often be suspected (Gaudier[14]).

The *treatment* consists in removal of the affected part. Resection

of the jaws need not be resorted to. However, the diagnosis must be verified before the extent of the operation is decided upon.

Syphilis of the Jaws.— Lues of the jaws, aside from primary infection of the mouth, appears in its tertiary form. The lesion usually takes on the form of circumscribed or diffuse osteitis and periostitis.

Circumscribed gummata appear most frequently in the hard palate. Primarily the lesion involves the mucosa of the roof of the mouth and extends to the bone. Here it causes a restricted area of swelling, which soon breaks down, ulcerates, discharges a few small sequestra, and perforates into the nares. In the lower jaw the infection is characterized by the formation of small nodules in the bone.

The *diffuse form of gummatous osteitis develops* at the alveolar processes in the form of a more or less painful swelling of the gums, which soon ulcerates and suppurates and is attended with necrosis of greater or lesser areas of the maxillae (see syphilis, p. 424).

The *diagnosis* is based on the presence of associated luetic lesions, the Wassermann reaction, and the primarily nodular character of the process.

The *treatment* of lues of the jaws, aside from constitutional medication, consists in extirpation of the local lesion.

For *suppurative osteomyelitis* see page 290.

For phosphorus necrosis see page 307.

Total Resection of the Superior Maxilla.— The mortality following resection of the jaw, the result of lung complications (aspiration pneumonitis), has led to the employment of *local anesthesia* in this class of cases with the view of leaving the "protective reflex activity of the larynx" unimpaired. Thus the patient expels the blood which flows into the air passages. The *operative field is made insensible* by abolishing the conductivity of the sensory nerves distributed to it. Five c.cm. of a 1 per cent novocain-adrenalin solution is used for the purpose in the following manner: The needle of the syringe is entered immediately below the angle of the malar bone and is advanced through the masseter muscle along the surface of the tuberosity of the maxilla a distance of from 5 to 6 cm., where it enters the pterygopalatine fossa. Contact with the nerve is indicated by the patient, who experiences a momentary sharp pain. The injection is then made and, as the needle is slowly withdrawn, a second 5 c.cm. is injected. This is followed by the abolishment of the sensory conductivity of the first division of the fifth nerve. The needle is entered just above the external angle of the eye and is carried along the bone to a depth of 4½ to 5 cm.,

where it reaches the superior orbital fissure and is arrested at the opposite side of the orbital wall. At this point 5 c.cm. of the solution is injected. A third, medial injection is made by entering the needle a finger's breadth above the inner angle of the eye and pushing it onward close to the bone, a distance of 4½ to 5 cm., where 5 c.cm. of the solution is injected; this is distributed over the medial wall of the orbit. In addition to this, the operative field, including the soft palate, is infiltrated with the same solution. When successfully carried out, the method is followed by complete anesthesia. The operative area is also rendered anemic (see Krause-Heymann,[15] Braun[16]).

When operating in full narcosis, the patient is placed in the Trendelenburg posture and the trachea occluded with the tampon tracheotomy tube, or peroral intratracheal narcosis is employed. Preliminary ligature of the external carotid artery is desirable.

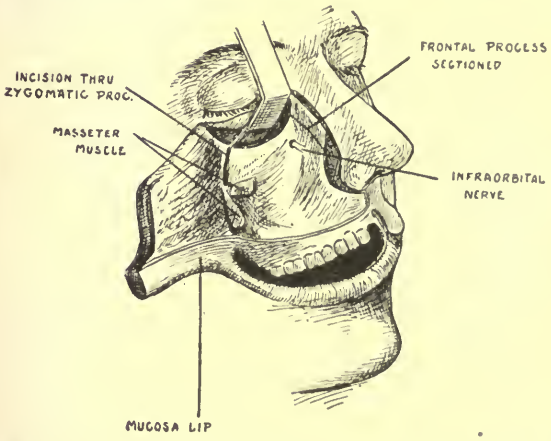

FIG. 801.— DIEFFENBACH-WEBER EXPOSURE OF UPPER JAW: LINES OF BONE SECTIONS.

The so-called Dieffenbach-Weber incision (Fig. 801), which avoids the facial nerve and Stenson's duct, is most frequently employed. After the soft parts are reflected outward, the masseter muscle is detached from the zygomatic arch, the ala nasi is pushed inward, and the orbital septum dissociated from the edge of the orbit. The contents of the orbital cavity are lifted up with a retractor. The following *bony connections* are divided: (1) the connection between the superior maxilla and the malar bone; (2) the connection between the frontal process and nasal bone; (3) the connection of the alveolar process and the hard palate with those of the opposite side; (4) the connection between the hard palate and the pterygoid process of the sphenoid bone. All of this is readily accomplished with the chisel. The operation is now transferred to the mouth. The internal incisor tooth is extracted; the mucoperiosteal covering of the hard palate is

sectioned in the median line, and the soft palate is cut close to the latter. The hard palate and the alveolar process are cut (with the chisel), also in the median line, and last the chisel cuts the connection between the maxilla and the pterygoid process of the sphenoid bone. The maxilla is grasped with a lion jaw forceps and twisted out. Temporary tamponade of the cavity effectually controls the bleeding.

For *excision of both maxillae* a similar technic is employed, the incision meeting in the median line of the upper lip. The nose is separated from the pyriform aperture and, after the cartilaginous septum is divided, is turned upward. Aside from the bone sections described above, the perpendicular plate of the sphenoid and the vomer are divided. An effort should be made to bring together the mucosa, with the view to shut off the nasal from the oral cavity. For modifi-

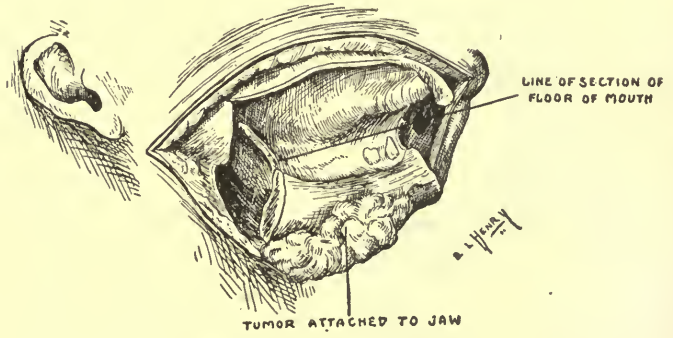

FIG. 802.— RESECTION OF THE LOWER JAW IN ITS CONTINUITY.

cations of the technic in this class of cases, see Krause-Heymann.[15] After the operation the patient is sat up in bed and the wound area is kept clean. Sterile nourishment is administered by means of a soft rubber catheter.

Most surgeons use a *provisional* prosthesis prepared from a model taken *before the operation.* This is introduced at once and makes the subsequent employment of permanent apparatus much easier. The object of the prosthesis is to enable the patient to speak and to masticate. It is astonishing what modern dentistry has accomplished in this connection.

Resection and Exarticulation of the Lower Jaw.— Resection of the lower jaw, without preservation of its continuity, is divided into:

1. Resection of the bone with solution of its continuity.
2. Exarticulation of one half of the jaw.

3. Total resection of the lower jaw.

Temporary resection of the lower jaw to afford access to the mouth is taken up elsewhere (p. 1679).

Resection of the lower jaw involves danger of aspiration pneumonitis and is followed by grave *functional disturbances* and serious *cosmetic defects.*

Resection of the lower jaw in its continuity begins with extraction of the teeth on either side of the doomed segment. The *incision* extends along the lower border of the chin or, which is simpler, the lip is split at its middle; this incision is carried to the center of the hyoid bone and the soft parts dissected back to the desired extent. The bone is divided with the Gigli saw and separated from its muscular attachments (digastric, mylohyoid, geniohyoglossus and geniohyoid) and the buccal mucosa. The tongue is kept from dropping backward by means of a silk suture. The bone is now removed and a previously prepared prosthesis or bone graft (Albee[17]) is placed in the gap. The mucosa of the mouth is sutured to that of the lip. Fig. 802 represents the conditions in removal of a portion of the body of the lower jaw.

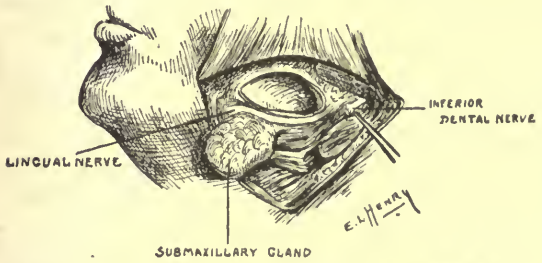

FIG. 803.— EXARTICULATION OF ONE HALF OF LOWER JAW.

Exarticulation of half the lower jaw is accomplished through a concave incision beginning at the center of the chin, skirting the hyoid bone, and ending at the mastoid process. The external carotid may be tied through this same approach. The flap thus formed is lifted upward, an incisor tooth is extracted at the point of contemplated bone section, the mucosa of the mouth is loosened from the bone, and the latter is cut with the Gigli saw. The anterior edge of the dissociated bone is grasped with lion jaw forceps and the jaw forcibly dislocated outward, putting the soft parts on a stretch at their inner aspect (mylohyoid, geniohyoid muscles, submaxillary gland and the internal pterygoid muscle) and dividing them close to the ramus. The jaw is still farther luxated and the tendon of the temporal muscle is divided at its insertion into the coronoid process. Disarticulation from the glenoid cavity is accomplished by forcible avulsion.

The problem of obviating deformity involves:

1. Secondary prosthesis.
2. Immediate prosthesis followed by secondary prosthesis.
3. Implantation of prostheses at the operation (ivory pegs, etc.).
4. Employment of a bone transplant from the patient.

Of these, the last is most desirable; the others belong to the highly specialized departments of dentistry (see Perthes[18] and his bibliography).

For fractures and dislocations of the lower jaw see Part IV.

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CHAPTER VIII

THE SURGERY OF THE NOSE AND ITS ACCESSORY SINUSES

Satisfactory examination of the nasal cavity is only possible when strong light is used. The rays of light must illuminate the objective on the same level as the vision of the examiner. This is readily accomplished by means of the light condenser and perforated head mirror so familiar even to the laity. The nares are held open by means of a nasal speculum which is sufficiently well known to justify omission of a description at this time. The introduction of a solution of cocain and adrenalin is indispensable to a satisfactory examination. Its use is objectionable only on the ground that it obliterates minor degrees of hypertrophy of the nasal mucosa. Refinements in this connection are described in special publications devoted to the subject (Coakley,[1] Ball,[2] Tilley[3]).

Malformation of the Nose.— Malformations of the nose and nasopharynx do not possess great surgical importance. Those of the *septum* are usually combined with other malformations. Perforation or total absence of the septum may be congenital, but is more likely to be due to perforating ulceration. *Incomplete development of the turbinated bones* is regarded as predisposing to *ozena*. Congenital coalescence of the inferior turbinated with the septum is not rare.

Congenital atresia of the choanae is usually fibrous in nature, though bony obstruction has been observed (v. Eicken[4]). It is easily corrected by puncture with suitable instruments, including a specially constructed trephine.

Injuries of the Nose.— Injuries of the nose in this connection are of importance only when they cause changes within the nasal cavity. This is especially likely to happen when the bones of the nose are fractured, i. e., the septum is "kinked" or fractured, or, what usually happens, the vomer is luxated from the quadrangular cartilage. Healing with deformity is the rule and is followed by more or less nasal obstruction on the convex side of the injured septum. If the *diagnosis* is made at once, the deformity may be corrected by tamponade of

the nostrils, and in severer cases, the nose is cocainized and the deformity is corrected by manipulation, and this is followed by appropriate tamponade. As injuries of this sort communicate with the nostril, infection is likely to occur. This may be prevented by cleanliness and frequent changing of the tampon.

Deviations and Spurs of the Septum.— Deformities of the septum occur most often at the junction of the *crista and spina*, where the vomer and quadrangular cartilage meet; in addition to this, they also appear as the result of *kinking of the cartilaginous septum,* due to trauma and, in a certain number of cases, the anterior portion of the latter deviates to one side without any assignable cause.

The *chief symptom* of the lesion is interference with nasal respiration; to this must be added certain reflex *disturbances,* such as asthma. In all cases the surgeon must assure himself that the obstruction is actually due to the septal deformity and not to the presence of another lesion, such as hypertrophy of the turbinated bodies or polypi.

The *treatment* varies with the degree of deformity. Small spurs or deviations may be removed with the chisel or other cutting instrument, provided the section does not perforate the opposite side. The grosser deformities are best treated by *submucous resection* (Killian[5]). A triangular flap, the anterior lower angle of which corresponds to a point just within the nares, is outlined on the mucosa of the septum and dissected up, together with the perichondrium and periosteum involving the deformity. The latter is cut off with cutting forceps or other instrument, care being taken not to invade the cavity on the opposite side. The flap is sutured back into place (see Nos. 1, 2 and 3 of bibliography).

Foreign Bodies in the Nose.— Of foreign bodies and rhinoliths, the former are most often introduced by children who force peas, shoe buttons, cherry pits and the like into the nostrils, where they lodge either in the nasal cavity or in the epipharynx. Unless the child is observed introducing the body, the subsequent clinical picture may be mistaken for that of infection of the accessory sinuses. Occasionally, a therapeutic agent (gauze, etc.) is retained in the nasal passages. Particles of cement, chrome, and arsenic may accumulate in the nostrils of persons working with these substances. In this way, the formation of *rhinoliths* is caused. However, these are also developed as the result of the incrustation of foreign bodies, clots of blood and inspissated mucus (Heymann[6]).

The *symptoms* provoked by the presence of foreign bodies and rhino-

liths consist in nasal obstruction and suppurative inflammation, headache and neuralgia. After a time, the inflammation becomes menacing and may extend to the accessory sinuses and the cranial contents, and, later still, the lesion takes on the form of a granulomatous process.

Removal of the offending agent is not difficult. In children, narcosis is desirable; in adults, cocainization of the nasal mucosa suffices. The obstructed passage is illuminated and the foreign body is gently removed with a slender, blunt hook. To prevent forcing the body into the narrow portion of the passage, the hook is introduced above it. If the body is large it should be crushed before removal.

Inflammations of the Accessory Nasal Cavities.— All of the accessory sinuses of the nose are susceptible to inflammation, either by extension from the nose or from other neighboring organs.

In processes of this sort, occurring in connection with general infection, it is not possible to be certain whether the excitants travel from the nose into the sinus or whether the infection is direct. It is certain that by far the largest number of cases of inflammation of the accessory nasal sinuses occur in connection with *influenza*. Of the other diseases, scarlatina is the most frequent associated general infection (Dmochowski[7]). Of the *local causes*, extension from the teeth (dental origin) is no doubt the most important, although *trauma* (especially by projectiles), *tuberculosis*, and *lues* are also responsible for the condition (Hajek[8]).

Acute Sinusitis.— Acute sinusitus, when not masked by the symptoms of the underlying general disease, presents the *clinical picture* of a septic infection. *Of the local symptoms, pain is the* dominant factor. The *pain* is very severe. For instance, in involvement of the maxillary sinus, the entire side of the face aches as in acute pulpitis dentalis. Pressure over the affected sinus increases the pain. In infection of the frontal sinus, pain is most severe over the eye. Infection of the ethmoid cells is often attended with ciliary pain and injection of the conjunctiva. Acute infection of the sphenoid cells cannot be diagnosticated. Occasionally, the skin or mucosa over the affected sinus is edematous. Progressive osteomyelitis in connection with acute sinusitis is very rare. Most cases of this sort are diagnosticated as neuralgia. The disease usually runs its course in two weeks. It is a regrettable fact that the affliction often escapes recognition even among well qualified practitioners.

The *diagnosis* is not easy. It rests entirely upon the location and character of the pain and the marked increase in nasal secretion.

Transillumination, unfortunately, is unreliable. When pus is present, the X-ray is of value. In doubtful cases, aspiration is of diagnostic and therapeutic value. One nostril is occluded and the air from the other is exhausted by means of a suction apparatus. Unless this is gradually done, pain is much increased for a time.

The *treatment* is not satisfactory. Pain is relieved by anodyne medication. Lavage of the affected sinus is contraindicated and, as a matter of fact, is too painful. Drainage of the sinus is permissible only when secretion is retained and provokes menacing symptoms. It is most often necessary in frontal sinusitis occurring in connection with scarlatina.

Chronic Sinusitis.— Chronic sinusitis is most often a sequel to an acute infection, though it is occasionally tuberculous in character.

The *dominant symptom* of chronic sinusitis consists in prolonged (though intermittent) discharge of pus from the nose. Certain postures of the head place the natural opening of the affected sinus at a low point so that the contents evacuated and the discharge from the nose ceases for a variable period of time. The total quantity of discharge is often very large, the patient soiling a dozen handkerchiefs a day. The character of the discharge is tenacious, slimy and of a white or yellow color. At times, it is green or even bluish, and if blood is present it is brown. When cheesy masses are discharged, the term *rhinitis caseosa* is used. Not infrequently the secretion dries within the nose in the form of crusts presenting a picture not unlike that of ozena. Depending upon the condition within the nose, the secretion is discharged anteriorly or flows into the pharnyx. In the latter event it gains access to the larynx where it gives rise to chronic irritation. As a rule, the discharge is associated with a fetid odor, especially when the lesion is located in the maxillary antrum.

Chronic sinusitis rarely heals without operation. On the other hand, fatal complications, such as meningitis, thrombophlebitis, etc., are rare. Acute exacerbations are not uncommon; spontaneous perforation into the orbit or through the canine fossa into the mouth is not infrequent. *Progressive osteomyelitis* of the walls of the affected sinus is a grave complication, especially of the frontal, in which event, the process gains access to the cranium and may be responsible for extradural or cerebral abscess and suppurative thrombophlebitis. Sequestration of the entire cranial vault has been observed in this connection. The treatment is taken up with the special sinuses.

Empyema of the Maxillary Antrum.— Empyema of the maxillary

antrum is the most common of the sinus infections. It must be diagnosticated from *osteomyelitis of the upper jaw.*

The *special symptoms* relate to the large quantity of the discharge and localization of the pain, which is most severe over the distribution of the second division of the trigeminus and in the teeth of the affected maxilla. The pain irradiates into the distribution of the supra-orbital nerve, but is less severe in this zone. Loss of smell is not constant but the patient is rarely aware of the odor which accompanies the disease.

The *diagnosis* is made by examination of the nose. When the nasal cavity is cleansed and pus is seen to emerge between the middle turbinated and the lateral wall of the nose, not from the fissure between the former and the septum, empyema of the sphenoid or posterior ethmoid cells may be excluded. If the secretion is ample, the anterior ethmoid cells may also be absolved. This reduces the possibilities to the frontal and maxillary sinuses, which is cleared up by placing a small tube into the ostium of the latter. If doubt still exists, the maxillary antrum may be punctured through the nose. After cocainizing the mucosa, the hollow needle is plunged into the antrum just above the inferior turbinated body. If necessary, the antrum may be emptied with a sterile salt solution (*diagnostic lavage*).

The *treatment,* at first, consists in frequent cleansing of the infected antrum through the ostium maxillare, a procedure which belongs to the domain of the specialist. If this fails, the antrum is opened; local anesthesia is usually all that is necessary.

Opening the maxillary antrum through the alveolar process is accomplished by extracting either the second bicuspid or the first molar tooth, after which the cavity is entered by means of a burr, a trephine or a large drill. The diameter of the hole should not be less than 5 mm. In making the opening the instrument must be directed slightly backward. The cavity is kept clean; between treatments the patient wears a prosthesis, prepared by the dentist, which prevents the entrance of food into the sinus and contracture of the drainage opening.

Opening of the maxillary antrum through the nose is credited to Mikulicz.[9] His method has been modified (Grant and Pegler[10]) and today is practiced as follows: The anterior one third of the inferior turbinated body is removed with cutting forceps and the antrum perforated at this situation with a specially constructed trocar (Fig. 804). The opening thus made is enlarged with the rongeur. The *after treat-*

ment is directed toward keeping the opening patent until the process heals.

Opening the maxillary antrum through the canine fossa is indicated in cases of chronic empyema in which prolonged drainage is necessary. Although the sinus was opened early in the last century (Desault[11]), the modern methods of accomplishing the purpose are based on the work of Küster.[12] An incision is made at the junction of the labial mucosa and alveolar process, extending from the canine tooth to the posterior molar; the mucosa and the periosteum are stripped up and the antrum is perforated with a burr or a gouge. This opening may be enlarged to any desired extent and is readily caused to communicate with one previously made from the nose (see above). The buccal flap of soft parts and another fashioned from the mucosa of the mouth may be turned into the sinus, a procedure which greatly facilitates ultimate obliteration of the cavity. Drainage and accessibility to cleansing manipulations are facilitated by the introduction into the dependent opening of a metal obturator (Fig. 805B).

Fig. 804.— Trocar for Perforating the Maxillary Antrum (Wilemnisky).

Empyema of the Frontal Sinus.— Empyema of the frontal sinus is most often the result of extension of infection from the nose, though it also develops in connection with general infections. It is often associated with a similar condition in the ethmoid and maxillary sinuses.

In the *treatment,* lavage of the sinus is practiced. This, however, is not often effective, in which event the next step consists in resection of the anterior portion of the middle turbinated body and opening the anterior ethmoid cells. If this is not followed by relief, the frontal sinus must be invaded from without. As simple external opening of the sinus is usually followed by the formation of a fistula, the modern method of treatment aims at obliteration of the sinus or establishment of a wide communication with the nasal cavity. As the former is not always successful, the operation most often practiced to-day is that of Killian,[13] which is a combination of the two. An incision down to the bone is made in the eyebrow area and is carried in a curve along the outer border of the nasal bone; externally, the incision extends to the end of the eyebrow. The soft parts and periosteum above the eyebrow are elevated in accord with the size of the sinus, which varies

widely and must be determined by X-ray before the operation. The sinus is opened with a gauge and chisel and its entire anterior wall is removed by means of bone cutting forceps. The mucosa is completely excised. Next, the skin at the lower aspect of the incision is turned down; the periosteum over the inner aspect of the orbital cavity is left undisturbed. The soft parts are dissected back from the frontal process of the superior maxilla, care being taken not to injure the tendon of the superior oblique muscle. Now the orbital portion of the sinus and the frontal process are carefully removed. The latter step exposes the frontal portion of the ethmoid cells and the anterior ethmoid labyrinth, which are converted into a single cavity. It now remains to establish communication between the frontal sinus and the nasal cavity. From the nasal mucosa, exposed by resection of the frontal process, a flap is fashioned the base of which is located downward; this flap is turned outward into the superficial wound, thus forming the desired wide communication with the nose, into which a drainage tube is introduced and the external wound is closed. The tube is removed on the third day. For additional methods see No. 8 of the bibliography.

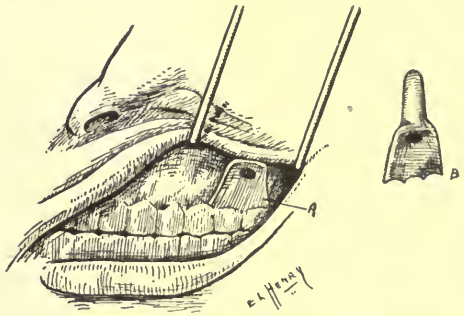

FIG. 805.— *A*, OPENING INTO THE MAXILLARY ANTRUM WITH OBTURATOR *in situ*. *B*, OBTURATOR.

EMPYEMA OF THE ETHMOID CELLS.— Isolated empyema of the ethmoid cells is very rare. They may be drained by resection of the middle turbinated body. From without they are invaded as a part of the operation for relief of infection of the frontal sinus (see above).

EMPYEMA OF THE SPHENOID CELLS.— Empyema of the sphenoid cells alone is not as rare as that of the ethmoid, but nevertheless, it occurs most frequently in combination with infection of one or more of the other accessory sinuses.

Aside from the *symptoms common* to multiple or pansinusitis, chronic infection of the sphenoid cells is characterized by the discharge of fetid material into the nose and the deposit of crusts. The clinical picture resembles that of ozena, and is, indeed, very often a complication (or perhaps a cause, Hajek[8]) of the latter condition. As a rule, the condition is attended with an atrophic, rather than a hyper-

trophic condition of the nasal mucosa. Of the subjective symptoms, disturbances of vision, i. e., scintillation and amblyopia, have been observed. While the connection is not definite, not a few rhinologists express themselves as convinced of an association of this sort (Holmes[14]).

Invasion of the sphenoid cells and their drainage may be accomplished from the nose without much difficulty, especially when the middle turbinated body is atrophied or is removed. However, the procedure belongs essentially to the field of the rhinologist. See bibliography, Nos. 1, 2, 3, and 8.

Ulceration and Infectious Granulomata.— Destructive and granulomatous processes are usually located at or near the entrance to the nares. Of these, simple perforating ulcer is the most common.

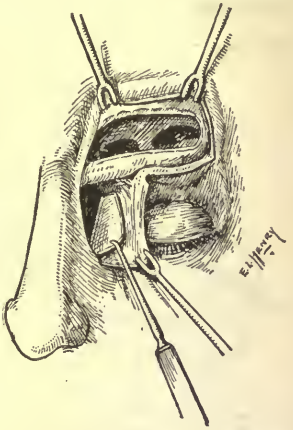

FIG. 806.— KILLIAN'S OPERATION FOR FRONTAL SINUSITIS.

The flap of nasal mucosa is held by a tenaculum.

SIMPLE PERFORATING ULCER.— Simple perforating ulcer is due to a chronic inflammatory process, a sort of seborrheic eczema, maintained by manipulations with the finger nail on the part of the patient. Is is usually seen in children, but it also occurs in adults who work in an atmosphere laden with arsenic dust, etc. In the latter class of cases considerable areas of the nasal cartilage become necrotic and the process is often attended with sharp hemorrhage. The *treatment* consists in removal of the cause.

TUBERCULOUS ULCERS.— Of the *specific ulcerations*, the tuberculous is the most common. It is most often associated with advanced lung tuberculosis, but may occur primarily in the nose, where it usually appears upon the cartilaginous portion of the septum, the lower turbinated body, or the floor of the nose. The ulcer bears some resemblance to lupus, but is likely to present a granulating surface; some of the granulations reach the size of a cherry. In not a few instances, multiple ulceration is present.

The *milder forms* of tuberculous ulceration of the nose heal after thorough curettement and the application of gauze saturated with 20 per cent pyrogallic acid. The *severer forms* are always associated with advanced lung lesions and are only susceptible to palliative measures.

Syphillitic Ulceration.— Syphilis is localized in the nose usually in the form of a tertiary manifestation, although chancre may also occur in this situation. In the latter instance the infection is no doubt carried by the finger nail.

In the *tertiary stage of the disease* the nose is usually ulcerated, though occasionally a syphilitic granuloma is found. The lesion occurs primarily as a gumma in the cartilage or bone, especially in the septum near the floor of the nose. Many of these perforate into the mouth and not a few destroy the bony framework of the nose. The *treatment* consists in energetic antiluetic medication, especially by the injection of mercury. Subsequently, the resultant deformity is subjected to plastic repair.

Granulomata of the nares occur in connection with lues. They usually arise from the turbinated bodies and are frequently multiple. Aside from those of luetic origin, tuberculosis, glanders, and lepra are the most common underlying causes of the peculiar infectious hyperplasia. The *diagnosis* is only possible with the aid of the microscope. Many writers include bleeding polypi and scleroma in the granulomata (see chapters devoted to lepra, scleroma, and glanders).

Nasal Hemorrhage.— In 80 per cent of the cases, nasal hemorrhage is due to simple ulcer and the polypoid growths developing in connection with it. This consideration suggests that an effort should be made to control the bleeding by firm anterior tamponade and that posterior tamponade should be postponed until it is shown that the former does not accomplish the purpose. It is, however, necessary that the anterior nares be thoroughly tamponed before the next step is taken. Of the various materials used for tamponade, there is none more effective than iodoform gauze. If the bleeding persists, the anterior tampon is removed, the nasal cavity washed with 2 per cent gelatin in normal salt solution, and the posterior nares tamponed by means of a Beloc cannula or, which is simpler and perhaps more desirable, by an ordinary soft rubber catheter passed through the bleeding nasal passage and "fished out" of the pharynx with the finger. To this the string to which the tampon is attached is fastened and delivered out of the nose as the catheter is withdrawn. Saturating the tampon with *thromboplastin* is of great value.

Benign Tumors of the Nose.— Fibromata of the nose are rare. As a rule so-called fibromata spring from the periosteum or perichondrium of the nose and consist of layers of connective tissue fibers. *Myxomata lymphangiectatica* are occasionally encountered. *Enchondromata*

are equally unusual. On the other hand, *osteomata* are not uncommon. They usually develop from the walls of the accessory sinuses and are often pedunculated. Tumors of this sort cause tremendous distortions of the face. The *diagnosis* is made with the X-ray.

The *operative removal* of large benign tumors of the nose is very difficult and involves sacrifice of the facial bones. The smaller intranasal fibromata or chondromata may be removed with the snare.

Nasal polypi are very common. The line of demarcation between these tumors and polypoid hypertrophies of the mucosa due to chronic inflammatory processes is not clearly defined. Most of the growths are pedunculated, though many of them are attached by a broad base. As a rule, they appear in multiple form; on the other hand, a solitary polypus may reach enormous dimensions and grow into the epipharnyx. The latter class of tumors often arises within the maxillary antrum.

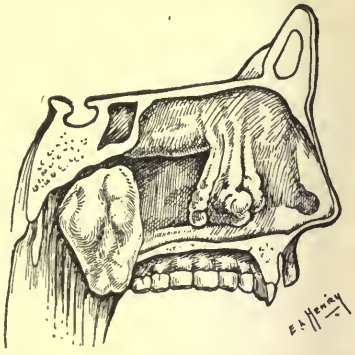

FIG. 807.— INTRANASAL POLYPI.

Ordinarily polypi are *benign in character* but are difficult to get rid of. The prevailing belief that they always recur is erroneous. If the growths are completely extirpated they do not return (Kümmel[15]).

The *dominant symptom* of nasal polypi is obstruction to breathing. To this may be added the discharge of a clear mucoid fluid from the nose. This leads to examination which readily reveals the nature of the lesion.

The *treatment* of typical polypi which do not originate in the accessory sinuses consists in intranasal extirpation with the *cold snare;* the older "polypus forceps" has been discarded. The nose is cocainized and illuminated and the snaring is accomplished "under the eye." When multiple polypi are present, the operative measure is repeated at intervals, until all the growths are removed. In polypi located in the epipharynx, application of the snare is difficult. In these instances the growths must be grasped with forceps and forcibly twisted out. If the polypoid growths are due to sinus infection, relief from the latter prevents recurrence of the former.

Malignant Tumors of the Nose.— *Sarcoma* originating within the nose is exceedingly rare (Heymann[6]).

Carcinoma of the nasal passages is usually of the horny, flat, epithe-

lial type. This is explained on the ground that the prolonged presence of an acute inflammatory process converts the ciliated epithelium into that of the epidermal type before the carcinoma develops. In the accessory sinuses carcinoma is of the cylindrical cell type.

The *symptoms* at first consist in nasal obstruction and deformations from pressure which occur early and increase rapidly. Soon the process takes on a destructive form, invading the contiguous sinuses, soft parts, and bones until the loss of substance is very extensive, including the eyeballs, the forehead and the bones of the cranium, often exposing the brain. Tumors originating in the accessory sinuses at first present the picture of sinusitis until the process gains access to the nasal passages. Involvement of the various cranial nerves is attended with pressure symptoms, and, later, with paralysis.

The *clinical picture* of malignant disease of the nose and its accessory sinuses is not distinctive until the process has advanved to a considerable extent, and the *differential diagnosis* is by no means easy. The presence of a tumor involves taking into account the large number of processes already taken up, of which benign tumors, tuberculosis, luetic lesions, granulomata, and polypi are of especial importance. The microscope is of course of the greatest assistance in this connection.

Preliminary Operative Exposure of the Interior of the Nose.—The development of rhinological technic has narrowed the field of operative exposure of the nasal passages to a considerable extent, and its usefulness is limited to making those tumors accessible which must be radically removed or which are of such dimensions as to render endonasal extirpation impracticable. Many of the methods available for the purpose are also used for approach to the nasopharynx. These are alluded to elsewhere. At this time the technic for exposure of the nasal passages alone will be given a brief consideration.

Section and separation of the soft parts of the nose in the median line, or the elevation of a wing of the nose at its insertion, does not give mueh room. It is rarely practiced at this time. Somewhat better exposure is attained by making an incision within the mouth, which loosens the upper lip, sectioning the septum and elevating the entire soft parts of the nose (Rouge[16]).

Section and Separation of the Entire Nose gives more room than the method just described (Koenig[17]). The soft parts of the nose are split from root to tip; the nasal bones are separated from the frontal process, the nasal process of the superior maxilla is divided,

and one half of the nose is displaced laterally. The opposite nasal passage may be exposed in a similar manner. The method most frequently employed is that of v. Bruns.[18] An incision is carried from the inner angle of the eye across to the opposite side and then around the nose through the septal attachment at the upper lip. The nasal bone and septum are divided in the line of the incision and the entire nose turned to the opposite side. This gives a fair exposure and, after the intranasal manipulations are completed, the nose is replaced and sutured. The cosmetic result is good.

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CHAPTER IX

THE SURGERY OF THE MOUTH

Malformations and Congenital Affections of the Mouth.— Branchiogenous clefts involving the mouth have already been taken up (p. 1599). This leaves for consideration *absence of the tongue, cleft tongue (lingua bifida), adhesions of the tongue, ankyloglossia, enlargement of the tongue, and epignathus.*

Absence of the tongue is usually associated with other defects, such as cleft palate (Griffith[1]).

Cleft tongue is the result of a failure of coalescence of the sides of this organ. It has been observed alone and in connection with congenital separation of the lower jaw. *Limitation of the deformity* to the tongue is fully described by Pooley.[2] It must not be confused with the deep furrow which at times is found in the tongue in tertiary lues.

Congenital pedunculation of the tongue is a peculiar soft enlargement of the anterior portion of this organ.

Adherent tongue is a term applied to a condition in which the lower surface of the tongue merges with the floor of the mouth, or in which the tongue is adherent to the sides of the lower jaw by bands of greater or lesser dimensions; not infrequently, the entire under surface of the tongue is "glued" to the floor of the mouth in this way. In these cases it is usually only necessary to separate the imprisoned organ from its restricting attachments with the finger. However, if any of the bands are particularly resisting, they may be divided with scissors. The normal mobility of the tongue prevents its readherence.

Ankyloglossia is a term applied to a condition in which the lower anterior aspect of the tongue is adherent to the floor of the mouth by a short broad frenulum ("tongue tied" of the laity). When the tongue is protruded it curves downward. These cases must show difficulty in nursing or speaking before they are interfered with. *Section of the frenulum is undesirable,* and fortunately is gradually being added to the list of forgotten operations.

Congenital enlargement of the tongue was first described by Petit

in 1742 (quoted by Girod[3]), who reported three cases in which the patients were in danger of suffocation from "swallowing the tongue." The condition would seem to be an increase of the parenchyma of the tongue (Welzel[4]). Lengemann[5] has practiced wedge shaped excision for relief of the affliction with success.

Epignathus, or protruding tongue, is usually a complication of other congenital malformations, such as encephalocele. It may be sufficiently marked to interfere with respiration. Resection of the tongue is not usually undertaken, because of the associated malformation.

Wounds, Burns, Scalds and Chemical Cauterization of the Buccal Mucosa.— Wounds of the buccal mucosa, that is, injuries restricted to this membrane, heal with astonishing rapidity, despite the active bacterial flora in the mouth. However, infection does occur in this situation, especially when the bone is injured. On the other hand, a wound produced elsewhere on the body by the teeth is very likely to be infected, which suggests that the mouth possesses a natural resistance to its "own bacteria," and that the latter possess considerable virulence in other environments. No doubt the great vascularity of the mouth presents an important defensive factor (Hunter[6]). Efforts at chemical sterilization of the buccal mucosa lessen its resistance to infection and, for this reason, mechanical cleansing is far more desirable. However, even the latter must not be attended with trauma.

The mouth is of course the seat of all kinds of wounds and no special classification is necessary in this connection. In deep wounds of the tongue, *hemorrhage* is the most important complication. This is attended with three possibilities, (1) exsanguination, (2) aspiration of blood into the lungs, (3) bloody infiltration of the parenchyma of the tongue. All of these may be obviated by *thorough hemostasis.* For the purpose, the tongue is delivered from the mouth, using a transfixion loop of silk if necessary. The employment of the suture ligature (p. 38) is especially useful in this situation. Persistent bleeding may demand *tracheotomy* in order to prevent asphyxiation, due to the flow of blood into the lungs. *Primary hemorrhage* is usually readily controlled; on the other hand, *secondary* bleeding is always a menacing condition, often controllable only by *ligature of the lingual artery in the neck* (p. 1029).

The *lodgment of foreign bodies* (fish bones, needles, etc.) in the tongue is a very frequent occurrence. They should be promptly removed under local anesthesia.

All FRESH WOUNDS OF THE TONGUE, INCLUDING THE CONTUSED AND LACERATED, SHOULD BE SUTURED, on the ground that the great vascularity of this organ and its resistance to infection makes uninterrupted repair a reasonable certainty. The fibrinous deposit seen on the wound a few days later does not interfere with healing; it corresponds to the scabs formed on superficial wounds situated elsewhere. *Partial avulsion or amputation of the tongue,* frequently due to injury from the teeth (fall or blow), is especially interesting in this connection, as replacement and suture of portions of the organ attached by even a slender pedicle are usually followed by healing. *Wasp stings* of the tongue are at times followed by such enormous swelling that respiration becomes seriously impaired, demanding relief by temporary tracheotomy. Return to the normal is the rule, though liberal incision, giving egress to the fluid exudate, may be necessary.

Ulceration of the tongue at the frenulum, due to prolonged friction against irregular teeth during an attack of *pertussis,* is often seen. The ulcer should be painted with 25 per cent silver nitrate, which is almost immediately neutralized with milk. This prevents infection which, when it occurs, is likely to extend very rapidly and has been known to destroy the entire tongue. Indeed, *decubital ulceration,* the outcome of friction against carious teeth, etc., should be given immediate attention. The condition heals at once when the cause is removed. So-called *Fede's*[7] *disease,* no doubt, belongs to this category.

Thermal burns of the buccal cavity occur in connection with the introduction of hot substances, usually fluid, and the entrance of hot vapors (see Pitt's[8] collection of cases). They possess especial interest because of involvement of the epiglottis, the air passages, the pharynx and the esophagus. These often demand *immediate tracheotomy.*

Pain in burns of the mouth is always severe, but soon disappears, except that deglutition remains painful. Cold (sterile) water affords considerable relief. Blebs rupture early. The epithelium soon assumes a grayish yellow color and insular areas form crusts, which soon exfoliate. If ulceration persists, these are treated as are benign ulcers.

Chemical cauterization follows the introduction into the mouth of corrosive substances (concentrated alkalies and acids). The lesion is likely to be extensive and is attended with much the same picture as heat produces, except that swelling and redness are likely to be marked. Involvement of the epiglottis, the pharynx and the esophagus is more frequent in these instances than in the thermal injuries. During the

repair the coalescence of the various structures of the mouth should be obviated as far as possible. Early plastic repair by sliding of the healthy mucosa is desirable.

Noma is taken up in Part II, chap. xii.

Lues of the Mouth.— Lues of the mouth is not exclusively a venereal disease. Of one hundred cases of non-venereal lues, collected by Münchheimer,[9] thirty-nine occurred in the mouth in the following order of frequency, lips, tongue, palate, cheek, gums. The carrier of the infection is very varied, including table silver, towels, handkerchiefs, etc. Direct infection (kissing, etc.) is probably the most common mode of contracting the disease.

The *primary lesion* does not differ from that located elsewhere in the body. When *lues is suspected,* the combination of the serological test, microscopical examination of a smear taken from the chancre, and the action of salvarsan should follow in sequence. In the mouth a papillary syphilid and the primary chancre may coexist (Fournier[10]).

Late forms of the disease possess the greatest surgical importance. Luetic lesions of the mouth appear as *circumscribed gummata* or in the form of *diffuse gummatous infiltrate.* Late lues of the *lip* usually begins in the corner of the mouth and gradually extends over its entirety. Gumma of the *cheek* perforates very early and takes on the form of a serpiginous ulcer.

Lues of the tongue, of the superficial and deep types, appears in the form of a nodule or nodules. Multiplicity of the lesion excludes actinomycosis and carcinoma. The deeper the gumma is located, the larger it is likely to be. The gummata grow slowly (size of a walnut) and are covered with apparently normal mucosa, until they break down which usually occurs in the center of the nodule. The subsequent behavior of the lesion is that of luetic *ulceration* (see Part II, chap. xxv). In the *differential diagnosis,* actinomycosis, tuberculosis, rhagades, and the ulcerative type of carcinoma must be taken into account. Aside from the constitutional *treatment* of luetic lesions of the mouth, curettage of the ulcers and excision of thickened areas are helpful measures (Morillon,[11] v. Mikulicz and Kümmel[12]).

Tuberculosis of the Mouth.—Primary buccal tuberculosis is rare. When one considers that the causative bacillus passes through this cavity *en route* to its lodgment within the body, this is an astonishing fact. No doubt the natural resistance of the mouth to infection,

spoken of above, is an important factor in this connection (Hildebrand[13]).

Secondary tuberculosis of the mouth develops in three ways, (1) *extension of lupus from the face,* (2) *the passage of tuberculous sputum from the lung,* and (3) *by way of the blood.*

The lesion appears in the form of a *single nodule* or of numerous *small, round ulcers.*

The *tuberous form* of conglomerate tubercles is almost always located at the edge of the tongue and closely resembles a gumma, beginning carcinoma, or an area of inflammatory thickening. Desirable as it is to recognize the character of the lesion at once, as a rule this can be ascertained (after removal of the offending process) only by the microscopical findings. It is true that the tubercle is very likely to be surrounded by a zone of inflammation. However, this is not determining as regards the diagnosis. The course of the lesion, after it breaks down, is more indicative. The secondary ulcer is characterized by *undermined edges,* thin rim, *"slit-like" form* and *grayish white base.* However, even these characteristics are not unlike those of a luetic lesion. Coexistence of laryngeal and pulmonary tuberculosis is so common that the mouth lesion might well be the outcome of an accidental complication.

FIG. 808.— TUBERCULOSIS OF THE TONGUE.

Tuberculous ulcerations of the mouth are exceedingly painful, especially when they develop secondarily to the nodular form. They interfere with the taking of nourishment — a most serious condition of affairs for obvious reasons.

In making the *diagnosis,* observations of the clinical course of the disease, the results of the v. Pirquet and the Wassermann tests, and the microscopical findings are taken into account. The coexistence of tuberculosis and lues has been shown by Garel and Armand.[14]

The *treatment,* aside from palliative measures to lessen pain, and

the employment of a non-irritating diet, consists in curettage and excision of the lesion. In advanced cases extirpation of the entire tongue must be considered.

Actinomycosis of the Mouth.— In most instances buccal actinomycosis is a part of a general involvement of the cheek, the maxillae, and the neck. As a rule, the *actinomycotic fungus gains access to this region through the mucosa of the mouth and the pharynx* (Illich[15]). The bacteriology and general clinical behavior of the disease is taken up in Part II, chap. xix.

Usually the primary port of entrance of the infection heals spontaneously; soon, however, the tissues at or near the site of entrance are involved in a board-like swelling, which presently breaks down in the form of more or less numerous abscesses, while the infection progressively invades the tissues in all directions. Thus, the cheek, the tongue, the floor of the mouth, the soft palate, and so on, are gradually involved and become the seat of necrotic areas and fistulae. The progress of the disease is essentially chronic in character.

The *diagnosis of actinomycosis* is made certain by recognition of the causative ray fungus. Unless this is done, it is difficult to diagnosticate the affection from lues, tuberculosis, and various tumors, including malignant diseases, at least not before the more menacing conditions have advanced sufficiently to render relief impossible.

For treatment see page 411. See also Latté and Barret,[16] Levy,[17] and No. 12 of the bibliography.

Phlegmonous Glossitis.— Superficial glossitis, attended with redness, epithelial exfoliation, papillary swelling, and fibrinous deposits, occurs in connection with almost all infectious diseases. The affection has been burdened with a number of fanciful names, such as lingua geographica, cat tongue, hair tongue, etc., the outcome of the imagination of the rhinolaryngologist. It possesses no surgical importance, except in as much as it forms a part of the more serious deep glossitis.

Deep glossitis (*glossitis profunda phlegmonosa*) may be the result of *trauma*. It occurs most frequently in connection with *erysipelas* of the mucosa of the mouth, the latter being invaded by extension from the face. This form of glossitis rarely suppurates.

A special form of glossitis — *hemiglossitis* — is characterized by a sudden swelling of one half the tongue. It runs a stormy course for three days and then subsides. The condition is ascribed by Güterbock[18] to nervous influences (analogous to herpes zoster).

So-called *mouth and hoof disease* (more or less widely distributed

among cattle) also belongs to the category of phlegmonous glossitis. The disease is epidemic and is preceded by a prodromal stage, indicating the development of an acute infectious disease. This is followed by the appearance on the tongue of a vesicular eruption, which also covers more or less of the mucosa of the rest of the mouth. In women, the eruption also appears on the nipples and on the vulva. Exfoliation of the blisters is followed by ulceration and this seems to provide an avenue of ingress for streptococci. In this event, the disease presents the clinical picture of a severe, deeply located phlegmonous glossitis. The mortality is high — 33 per cent (Siegel[19]).

Suppurative phlegmonous glossitis, despite the many injuries to which the tongue is subjected, is comparatively rare. When the infection is limited to the preëpiglottic region, deglutition is painful and this causes the condition to be mistaken for angina. When an abscess develops in this situation, the symptom of fever, together with swelling of the cervical *lymph nodes,* is presented. As the tongue lesion increases, the epiglottis is very likely to be involved, so that respiration becomes seriously embarrassed and, in not a few instances, tracheotomy is imperative. In the milder forms of the affection the disturbances are limited to pain and limitation of the mobility of the tongue. In the more severe forms of glossitis the entire tongue swells and may be so large as to protrude from the mouth. In these instances the tongue is forced between the teeth, and presses the floor of the mouth downward and backward against the palate and the larynx, causing *respiratory difficulty without the occurrence of edema glottidis.* Of course, speech and deglutition are impossible.

The *dangers* of the condition are readily understood. Aside from the *menace as regards respiration,* the process is very likely to extend from the floor of the mouth and to *descend into the neck,* while Ludwig's angina usually begins with a periodontitis, the disease infects the floor of the mouth before descending in the neck, from where it invades the loose alveolar tissue in the region of the submaxillary triangle and usually does not let the tongue escape entirely. This would indicate that perhaps so-called Ludwig's angina is not as clearly defined a clinical entity as has been supposed — a notion the writer is inclined to share. The last, but not by any means the least, danger of phlegmonous glossitis is that of *aspiration pneumonitis.*

The *treatment* of phlegmonous glossitis must be radical. Despite the attendant pain, the finger is forced through the angle of the mouth and is "hooked" behind the base of the tongue. Under the guide of

the finger a scalpel is introduced (sideways) as far back as possible, its cutting edge turned downward and the dorsum of the tongue deeply incised as the knife is withdrawn. The incision is made in the median line unless one side of the tongue is more swollen than the other, when the incision is made through the zone of greatest swelling. Even though pus is not discharged, the incision gives exit to sufficient exudate to afford relief. When healing occurs, the resultant scar is barely perceptible. Bleeding, unless menacing, is encouraged; if this becomes alarming, tamponade readily accomplishes hemostasis. If respiration is embarrassed, *tracheotomy* is performed before the liberating incision is made. If no contraindication exists, tracheal narcosis may then be employed and the tongue incised with less haste. If the infiltration has invaded the submental or submaxillary regions, these must also be freely incised. As may be expected, the vascularity of the tongue renders gangrene a rare occurrence.

Glossitis hemorrhagica consists in a rare bloody extravasation into the parenchyma of the tongue occurring in connection with hemophilia. The tongue may become so distended that, despite the danger of persistent bleeding, it must be incised (Umbreit[20]).

Hemangiomata of the Mouth.— Simple hemangiomata (telangiectases) appear on the tongue in the form of small, slightly elevated spots, somewhat darker in color than the surrounding normal mucosa. They are usually associated with a similar condition in the mucosa of the cheek, though the latter are, as a rule, larger in size. In most instances, the simple form of the lesion is superimposed upon an underlying hemangioma cavernosum situated in the submucous tissues. The tumors always increase in size when the head is lowered and they may be emptied by pressure; as soon as the pressure is released the dilated vessels are again filled up (see Part VI, chap. x).

The *clinical importance* of hemangiomata lies in their tendency to extend in all directions. They often assume gigantic proportions. The very objectionable unsightliness of the affliction is the least important factor in the problem. Tumors of this sort are very likely to *bleed in large quantities and readily infect,* and therefore are dangerous to life.

Hemangiomata should, when possible, be *extirpated.* The technic of the procedure does not differ from that employed for the removal of other growths in this situation. For *ignipuncture, injections of alcohol,* and the use of magnesium pencils see page 1163.

Lymphangiomata of the Mouth.— In the mouth, lymphangiomata

appear in the *cystic and cavernous forms.* Cystic lymphangiectasis appears as simple cysts which correspond to dilated lymph vessels, are lined with a single layer of epithelium, and contain a serous fluid. They are probably retention cysts and may reach a considerable size. In addition to this, cystic lymphangiomata appear in the form of *warts and nodules,* either disseminated over the entire tongue or collected in groups at the side and on its edge. The cysts usually rest upon a base of indurated tissue, which is the outcome of a low grade of inflammation.

Both forms of the lesion are congenital in origin, but grow so slowly that they do not attract attention until they achieve a certain size or become inflamed. The latter is always due to an accidental infection (Küttner[21]). This may assume alarming proportions and present the clinical picture of a phlegmonous glossitis.

Fig. 809.— Macroglossia.

The smaller cysts and nodules may be *destroyed with the cautery;* the larger ones are best *extirpated.*

Macroglossia.— Cavernous angioma of the cheek is called macromelia; of the lip, macrocheilia; and when the tongue is involved the term macroglossia is employed.

Macroglossia is congenital in origin, but usually does not attract attention until the tongue protrudes beyond the teeth. The protruding portion of the tongue is dark in color and covered with fissures, excoriations, and ulcers. The intrabuccal part of the organ is indented by the pressure of the teeth and alveolar processes. If the dorsum of this portion is visible, it is found covered with numerous vesicles. The process may cause little or no disturbance for years and then, suddenly, a rapid increase in size or an intercurrent inflammation causes the tongue to become a great hindrance to function or, if the infection is severe, may menace life in a manner described in connection with phlegmonous glossitis.

Histologically the process does not differ from lymphangiomata in-

volving other portions of the body. Muscular and neurofibromatous macroglossia are very rare.

As a rule, macroglossia affects only the anterior portion of the tongue. It may also be restricted to certain areas of this organ and simulate other forms of growths.

The *treatment* of the smaller cavernous lymphomata consists in destruction by the cautery or by excision. In the severe forms of lymphangiomatous macroglossia repeated wedge shaped excision of the enlarged tongue is practiced. Most surgeons precede the primary operation by ligature of both lingual arteries, a measure which simplifies the subsequent problem considerably.

Mucous Cysts of the Mouth.— Mucous cysts appear on the mucous surfaces of the lips and cheeks and occasionally on the edge of the tongue. The tumors are round in form, fluctuate, and are often bluish in color. They contain a clear, ropy mucus and belong to the class of *retention cysts of the mucous glands.*

In making the *diagnosis,* cysts of this nature must not be mistaken for hemangiomata. The *treatment* consists in extirpation which is readily accomplished under local anesthesia.

Cysts of the back of the tongue in the region of the foramen cecum, the so-called lingual tonsils in front of the epiglottis, possess great surgical significance. They originate in the small mucous glands of the crypts of the tongue, in the larger mucous glands between the papillae, and in the remains of the thyroglossal duct. The latter form is lined with ciliated epithelium (Schmidt[22]). Cysts of this nature irritate the epiglottis and are often responsible for persistent cough. They are discovered by examination with the mirror and are easily extirpated with appropriately fashioned scissors.

Echinococcus cysts of the mouth are exceedingly rare.

Ranula.— The term *ranula* (batrachos hypoglossis, frog swelling, grenouillette of the French) is applied to a sublingual cyst which occupies the space between the frenulum and the mental portion of the lower jaw, pushing the tongue upward. The cyst usually develops on one side and gradually crosses the median line. Its outline is made asymmetrical by the constriction of the frenulum. The large cysts bulge beneath the jaw. Occasionally, a ranula develops in the median line.

As a rule, Wharton's duct may be probed as it lies on the top of the transparent cystic enlargement (Fig. 810). The cyst itself appears as a transparent protrusion, grayish or bluish red in color, with the blood

vessels distinctly visible in the superimposed mucosa of the floor of the mouth. The contents of the cyst is visible shimmering through its covering and conveys the idea of a blister. As a rule, the cyst is unilocular, though occasionally it is divided into multiloculi. The fluid does not contain ptyalin or *rhodan kalium.*

Ranula occurs at all times of life and may be congenital. Its growth is slow but steady, so that the *disturbances,* which at first are minor, may, and indeed often do, assume considerable proportions. These consist in interference with nourishment, and ultimately with respiration. When infection occurs, which is by no means rare, the clinical picture assumes that described in connection with phlegmonous glossitis, especially its complications. If the ranula develops in childhood, its presence interferes with development of the jaw (atrophy).

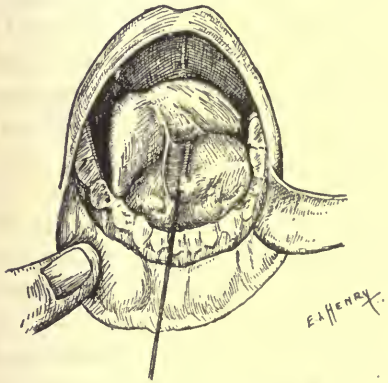

FIG. 810.—RANULA: PROBE IN WHARTON'S DUCT.

The presentations of the *pathogenesis of ranula* have been so varied, that the views held at this time may properly be stated briefly as follows: (1) that it is a cyst consisting of dilated Bochdalek's tubules lined with ciliated epithelium, which probably is correct (Neumann[23]); (2) that is is a dilatation of the Blandin-Nuhn gland, which probably is not correct (v. Recklinghausen[24]); (3) that it is a chronic inflammation of the sublingual gland, which probably is correct (v. Hippel,[25] Morestin[26]). The theory that ranula is a retention cyst of the sublingual gland has been abandoned.

The *differential diagnosis* takes into account tumors of various kinds located in this situation. Though solid growths should be easily differentiated, cysts of other kinds than ranula are not so readily separated, nor is confusion with *angiomata* and *lymphangiomata* always easily avoided. However, attention to detail in studying the characteristics of the growth should prevent an erroneous conclusion, except perhaps with respect to dermoids of the floor of the mouth, in which the character of the contents clears up the situation.

The *treatment* consists in *extirpation* of the cyst through the mouth. This may usually be accomplished under novocain-adrenalin local anes-

thesia. When, however, the cyst is a part of the sublingual gland, the latter is best excised *in toto* through an incision beneath the jaw.

Dermoids of the Mouth.— In 1908 Debonelle[27] collected seventy-seven cases of dermoid cysts of the mouth. They are the outcome of inversion of the outer layer of the second and third, and in some instances that of the first with the second, branchial folds (see Dermoids, Part VII, chap. i). As a matter of fact, most dermoids in this area are found in the subhyoid and submental regions between the skin and the floor of the mouth, where they are intimately connected with either the hyoid bone or the inferior maxilla. Most dermoids are separated from the floor of the mouth by the geniohyoid and the geniohyoglossus muscles (submental dermoids), although at times they are found located between the mucosa and the muscles mentioned (*sublingual dermoid*).

The *submental dermoid* presents, at first, below the chin, is covered with normal skin, and is elastic in consistence. As the tumor increases in size it elevates the floor of the mouth and then bears a resemblance to the sublingual form.

The *sublingual dermoid,* at first, is not palpable below the chin. As it grows it protrudes somewhat in the latter situation, but never to the extent that obtains in connection with its allied growth, i. e., the submental form. When it reaches a certain size it cannot be differentiated from ranula (see above).

The *treatment* consists in extirpation of the tumor. Irrespective of whether the dermoid is sublingual or submental, it is best removed from without, a maneuver which does not include entering the mouth (v. Hippel[25]).

The rare teratoids of this region are extensively discussed by Niemczyk,[28] Reuter,[29] Rolando,[30] and Blanc.[31]

Solid Non-carcinomatous Tumors of the Tongue.— Lipomata of the mouth, and especially of the tongue, are not common. They usually spring from the submucosa or between the layers of muscles. They possess all the characteristics of a fatty tumor. Multiple lipoma of the tongue is reported by Malon[32] (see also bibliography of No. 12, which is very extensive). Extirpation of the growth presents no difficulties.

Connective tissue tumors, *fibromata, neurofibromata and fibromata with cartilaginous centers,* are occasionally found in the tongue. It is not possible to recognize the exact character of these growths. However, the clinical picture permits the conclusion that a benign process is being dealt with.

Papillomata of the mouth are usually pedunculated and are found on the uvula, the pillars of the fauces, and the dorsum of the tongue. As a rule they are about the size of a pea or a bean, but occasionally they grow to that of a hazel nut. Tumors of this sort are usually discovered during an examination of the mouth made for another purpose. They are readily removed with the snare or scissors.

Endotheliomata of the mouth are exceedingly rare. They are most often found lodged in the palate close to the cheek, i. e., the arcus palatoglossus. Tumors of this sort present the clinical picture of a benign process, the character of which can be determined only by the microscope.

Adenomata are usually situated at the base of the tongue. A tumor consisting of *thyroid tissue* is also occasionally found in this location (Chamisso de Boncourt[33]). The latter is, no doubt, the outcome of an atypical embryonic development. The removal of the growth is likely to be attended with great difficulty. In most instances it will be found necessary to proceed as in excision of the tongue for carcinoma (p. 1677).

Sarcomata of the mouth possess the peculiarity that they often exist for years without causing disturbance and then suddenly rapidly increase in size. In these instances the tumor breaks down very early and presents the clinical picture of malignant disease. The exact character of the process can be determined only by the microscope. Of course, immediate removal of the growth and of as much of the contiguous normal tissue as is possible is imperative (Marion,[34] Melchior[35]).

Carcinoma of the Mouth.— Carcinoma in the mouth originates in three situations, the tongue, the cheek, and the mucosa of the floor of the mouth. The story of its *causation* would seem to be staged in an environment subject to chronic irritation, and to the "tissue hyperplasia" (precancerous stage of Hutcheson) that goes with the latter. Of these processes, *leukoplakia* is the most dominantly determining. (For statistics in this connection see v. Bergmann-Küttner.[36]) The influence of lues upon the subsequent development of carcinoma of the tongue is not decided, yet the affirmative view is strongly supported (Butlin,[37] Warren,[38] Poirier[39] and their bibliographies).

Carcinoma of the tongue is practically always of the pavement epithelial type. Clinically the lesion may be divided into *superficial and deep*. The former begins in the surface epithelium, the latter, in the glands of the mucosa. Primarily, it appears in the form of a nodule,

but this soon breaks down and an ulcer is formed, that is, carcinoma of the tongue may be said to present a *tumor and an ulcer stage.* The characteristics of both have been taken up *in extensio* in Part VI, chap. xvii.

In carcinoma of the tongue the *lymph nodes of the neck are involved early in the process.* The lymphatics of the entire tongue communicate very freely with one another, which accounts for the involvement of the lymph nodes of the neck on the side opposite to the tongue lesion (Küttner[36]). In the neck the carcinomatous infiltrate invades, primarily, the lymph nodes in the submaxillary and the submental regions (not the salivary glands) and those surrounding the internal jugular vein. In addition to this, the nodes at the tip of the parotid are promptly invaded. When the process extends far beyond the neighborhood of the mouth, as it often does, the condition of the patient is nothing short of horrible. Strange to say, despite the rapid lymphatic extension of carcinoma of the tongue, the formation of metastases is comparatively rare.

The *diagnosis,* as a rule, is not difficult, especially when ulceration is present. However, serious errors are frequently made. The most common of these is the assumption that the process is luetic in origin. Delay in differentiation from pressure ulceration, actinomycosis and tuberculous processes is also a common occurrence. When the dangers involved in allowing a carcinoma to remain are considered, the removal of a gumma of the tongue is not a reprehensible act. The accessibility of the process in question to the removal of a section for microscopical examination makes omission of this helpful measure unforgivable. In each instance a complete history of the behavior of the growth must be obtained, and when this, together with a careful study of its physical characteristics and the microscopical findings, is analyzed, error in diagnosis will be avoided. There is no greater fallacy than to assume that a "positive Wassermann" decides the character of a lingual ulcerative process. On the contrary, while a causative relationship between lues and carcinoma is not proved, *chronic inflammatory processes unquestionably present a condition of affairs favorable to the development of carcinoma.*

Carcinoma of the floor of the mouth presents a picture much like that of the tongue. It usually begins at the frenulum and extends along the body of the jaw, which it promptly invades. Once it attains certain dimensions, its subsequent behavior is in every regard similar to that of the tongue.

Carcinoma of the mucosa of the cheek usually begins in the fold where this membrane is reflected over the ascending ramus of the jaw. The nodular stage is brief and ulceration follows rapidly. *Carcinoma of the mucosa of the hard palate* soon invades the underlying bone. The process in these situations is similar in every respect to that involving the tongue.

The special consideration with regard to the *surgical treatment of carcinoma of the tongue,* aside from extirpation of the growth, relates to removal of the lymph nodes from both sides of the neck by one of the so-called *typical methods.* Of these, that of Küttner[36] (Fig. 811) gives the widest exposure (for the other incisions see Krause-Heymann[40]). The method permits of unilateral or bilateral ligature of the lingual and external carotid arteries. When it is necessary to section the jaw, this is postponed for a few days to avoid extension of the infection into the neck wound from the mouth. Removal of the submaxillary salivary glands renders the deep lymph nodes in this situation more accessible.

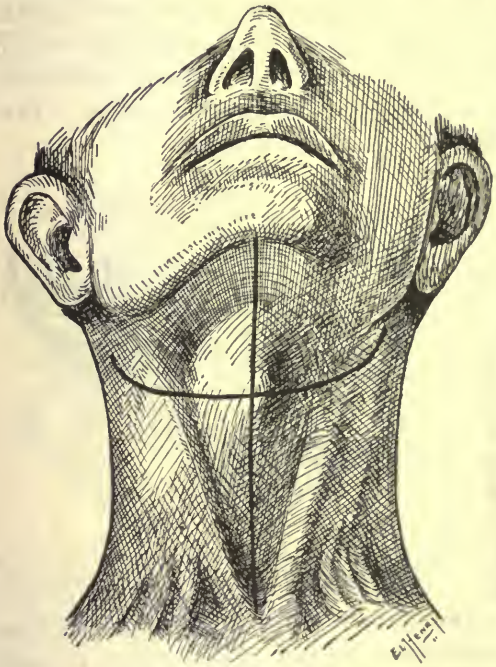

FIG. 811.— KÜTTNER'S INCISIONS FOR REMOVAL OF THE CERVICAL LYMPH NODES IN CARCINOMA OF THE TONGUE.

The next step in the procedure is the radical *removal of the tumor of the tongue.* This may be accomplished under local anesthesia. When narcosis is employed, the patient is postured so that blood does not gain access to the air passages. In all extensive operations upon the tongue, *bilateral ligature of the lingual arteries is imperative.* If the tissues contiguous to the tongue must be excised, unilateral or bilateral ligature of the external carotid artery is justified. The facial arteries are ligated when the submaxillary glands are removed.

Butlin[37] practices preliminary laryngotomy (70 cases) and administers narcosis in this way. This is a very useful measure.

For the purpose of surgical efforts at relief, the tongue may be reached as follows:

1. *Through the mouth* the greater part of the tongue may be removed with less difficulty than is generally believed. In 1908, Butlin[37] reported 197 cases approached in this way. In none of these did he deem it necessary to divide the jaw, although in a few instances he excised a portion of the latter. For the purpose, the lower maxilla is widely separated from the upper by means of the Whitehead mouth gag (Fig. 774). The tongue is grasped with forceps or a traction suture is introduced. As the frenulum is divided the tongue is progressively delivered from the oral cavity, and sectioned as far back as is possible. As the result of preliminary ligature of the lingual arteries (which must always be done), the bleeding is inconsiderable, and is readily controlled by the usual means. As preliminary exclusion of the stump from the circulation favors gangrene, it is not sutured.

Fig. 812.— Incision for Exposure of Base of Tongue and Palate (v. Bergmann).

2. *Section of the cheek* gives little additional room and is employed only when the growth is small and located at the posterior portion of the edge of the tongue. The incision is made below the level of Stenson's duct.

3. *Submental* (*suprahyoid*) or *submaxillary* incisions give opportunity for the removal of a certain number of lymph nodes. A transverse incision is made just above the hyoid bone, and this is supplemented by a second vertical section as far as the middle of the larynx. The muscles attached to the lower jaw are divided and the mucosa of the mouth is opened. Through this opening the tongue is delivered and amputated. The method is used only when the carcinoma is limited to the anterior portion of the tongue and the floor of the mouth immediately contiguous to it.

Kocher's[41] *incision* begins at the middle of the lower jaw, extends

to midway between the chin and hyoid bone, then parallels the body of the inferior maxilla to the anterior border of the sternomastoid muscle and thence backward and upward to the lobule of the ear. The flap is turned up and the lymph nodes of this region, together with the salivary maxillary gland, are removed, exposing the lingual artery, which is tied. Next, the mylohyoid muscle and the mucosa of the mouth is sectioned; the tongue is delivered through the wound and amputated.

4. When the carcinoma involves the base of the tongue, or the palate and floor of the mouth, *the lower jaw must be sectioned.* Von Bergmann's[42] modification of v. Langenbeck's[43] method is largely practiced today. The cheek is divided transversely downward to the mucous membrane, from the angle of the mouth to the anterior edge of the masseter muscle, then downward at an acute angle, dividing all the soft parts, to the submaxillary region (Fig. 812). The jaw, corresponding to the latter incision, is freed of its soft parts and divided with a Gigli saw in an oblique direction (obliquely downward and forward), which facilitates subsequent apposition of the fragments. When the mucosa of the mouth is divided, the two ends of the separated bone can be held apart readily with retractors, and the submaxillary tissues conveniently extirpated. The approach to the oral cavity and to the lateral and posterior aspects of the pharynx is now satisfactory and as much of these structures may be removed as is necessary (Fig. 813). The method permits exposure of the facial and lingual arteries, which may be ligated before division. Contamination of the wound from the mouth is best guarded against by suturing the mucosa of the pharynx to the lower angle of the skin wound, where it lies close to the larynx. The secretions are thus directed into the dressing. The bone fragments are held in apposition with wire.

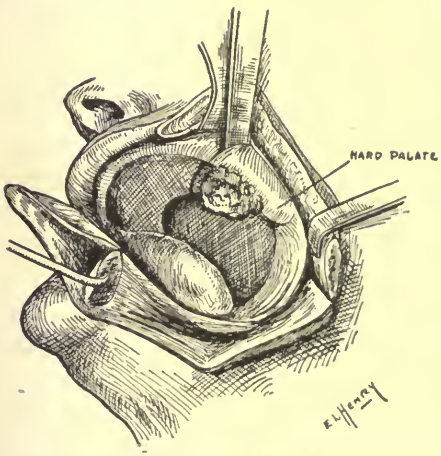

FIG. 813.—EXPOSURE OF MOUTH AND PHARYNX THROUGH THE V. BERGMANN INCISION SHOWN IN FIG. 812, HARD PALATE BEING CHISELED AWAY.

5. *Resection of a portion of the lower jaw,* together with extirpation

of more or less of the oral contents, may be accomplished by the v. Bergmann method just described. By this method removal of the ramus of the inferior maxilla is easily accomplished. In totaling the cases of a number of surgeons, Küttner[36] estimates the mortality attendant upon extensive extirpation of carcinoma of the tongue at about 16.2 per cent.

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CHAPTER X

SURGERY OF THE PHARYNX

Malformations of the Pharynx.— Of the malformations of the pharynx, few possess surgical interest. *Teratoma* is occasionally found in this situation. *Epignathus,* because of the interference with breathing which it causes, soon destroys the host. However in several instances the mass has been successfully removed.

Diverticula, cysts, and fistulae are due to errors in development of the branchial clefts. Diverticula of the upper branchial cleft are located near the eustachian tube; those of the second, correspond to a dilated fossa of Rosenmüller, or they may be of sufficient size to extend to the skin. Diverticula on the ventral side of the second branchial cleft, from which the tonsillar fossa is derived, are the most common, although not so common as are fistulae in this situation. Diverticula in this region are likely to be very extensive, encroaching upon the anterior triangle of the neck between the sternomastoid and the trachea. Diverticula of the third branchial cleft are located above the superior laryngeal nerve and protrude between the hyoid bone and the thyroid cartilage.

Congenital cysts appear in all locations in which diverticula are found. To this class belongs the cyst found in the sinus pyriformis, and probably also those found in front of the epiglottis and in the glossopharyngeal ligament.

Branchiogenous cysts of this sort are at times responsible for a peculiar metallic quality of the voice of the host, and are very likely to interfere with deglutition. They should be extirpated by one of the external methods discussed farther on (Wheeler,[1] Schneider,[2] Kümmel[3]).

Inflammatory Diseases.— Inflammatory diseases of the pharynx and of the tonsils, especially diphtheria, are taken up in the general part of this work.

Phlegmonous Tonsillitis and Peritonsillitis.— Phlegmonous inflammation of the tonsil and the surrounding tissues furnishes a clini-

cal picture of some surgical importance. Suppurative processes in the tonsil always develop in connection with peritonsillar phlegmon, which renders establishment of the original seat of the infection uncertain. The chronic form of tonsillar abscess (so-called) corresponds to a dilated tonsillar crypt filled with purulent secretion and detritus.

Peritonsillar phlegmon is characterized by an inflammatory swelling of the capsule of the tonsil, which forces apart the pillars of the fauces. The inflammatory infiltrate in the peritonsillar tissue is dominantly manifest at the upper aspect of the palate, where the lymphatics are most abundant. In severe cases the infiltrate extends to the lymph nodes at the angle of the jaw and into the retropharyngeal connective tissue.

The *onset of a phlegmonous tonsillitis* is usually heralded by a chill and a sharp rise of temperature, which is associated with an acute infectious process. This is soon followed by dysphagia and more or less localized pain at the seat of the infection. If the process persists for three or four days, the uvula is also infiltrated, and in not a few cases invasion of the epiglottis is responsible for serious interference with respiration, so that tracheotomy becomes necessary.

Spontaneous rupture of the abscess occurs into the supratonsillar fossa, occasionally through the anterior, and, in rare instances, through the posterior pillar of the fauces. Death may be caused by the sudden rupture of an abscess of this sort and the discharge of pus into the larynx. Fatal results have been the ouctome of ulceration into the internal carotid artery, extension of the infiammation into the mediastinum, extension of a secondary suppurative phlebitis into the jugular vein, and of a general infection.

The *diagnosis* is not difficult. However, unless the throat is examined, the condition will escape recognition. As opening the mouth is difficult, the surgeon must depend upon the palpating finger in this connection. Discovery of the infiltrate, together with the picture of an acute infectious process, should make the diagnosis. Difficulties arise only in the milder forms of the disease, when the question of the presence of a new growth, complicated by a local inflammatory reaction comes up. In these instances aspiration of the area of thickening should be resorted to.

The *treatment* consists in incising the inflamed area. This must not be too long delayed. In making the *incision,* the rule laid down by Chiari[4] may be followed. The knife is entered at a point midway between the base of the uvula and the posterior molar tooth, where

is may be advanced to a depth of from 1 to 2 cm. without danger. It is advantageous to increase the size of the opening with the Mayo scissors (Fig. 510). Local anesthesia is not desirable. As phlegmonous peritonsillitis is an exhausting disease, nourishing the patient through a nasal catheter is a valuable measure. After the process has subsided, the question of removal of the tonsils must be taken up.

The Retropharyngeal Abscess.— Abscess behind the posterior wall of the pharynx results from *gravitation* of an infection of the cervical vertebrae, of the base of the skull, or from extension of an infection by means of the *lymphatics* situated in its posterior wall. The lymphatics in this situation are very numerous and drain the nose, its accessory sinuses, the posterior wall of the pharynx and the pharyngeal tonsils, any of which may be the port of entrance of the causative excitants.

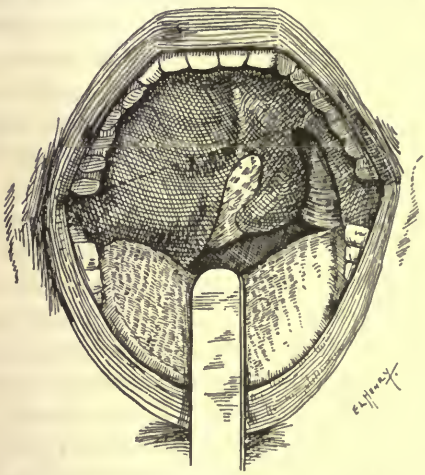

Fig. 814.— Peritonsillar Abscess. Showing point where liberating incision should be made.

The *clinical picture* of retropharyngeal abscess is that of a general septic infection, together with disturbances of deglutition and respiration. The latter is caused by mechanical obstruction and by inflammatory edema of the tissues. Both of these may reach extensive proportions and often threaten the life of the patient. Rupture of the abscess involves the danger of death by strangulation. This is especially true in childhen in whom the lesion is most frequently seen.

The *diagnosis* is not made as often as it should be. It rests mostly upon the examination which reveals the presence of a mass, usually covered with almost normal mucosa, causing the posterior pharyngeal wall to bulge. Palpation elicits fluctuation which, together with the general symptoms of infection, should make the diagnosis certain.

The *treatment* consists in opening the abscess in the median line with the patient's head lowered. When the abscess is tuberculous (in connection with spondylitis) it is aspirated and injected with iodoform emulsion.

Hypertrophy of the Pharyngeal Tonsil.— Hypertrophy of the pharyngeal tonsil (erroneously designated as adenoid vegetation), in children up to twenty years of age, is the most frequent cause of interference with nasal respiration and of acute and chronic middle ear infections; it is, therefore, a lesion of great surgical importance.

While the *etiology of hypertrophy of the pharyngeal tonsil* is unknown, it may be definitely stated that the lesion is not tuberculous, nor does it constitute a port of entrance for this disease.

Objectively, the presence of the affection may be established by anterior or posterior rhinoscopy or by palpation. Obstruction to nasal breathing is the most striking of the *subjective symptoms.* To this is added mouth breathing, a stupid expression of countenance, and disturbances due to irritation of the air passages. If relief is not afforded before the second teeth appear, permanent deformation of the maxillae often occurs. Interference with the normal processes of life caused by obstruction to nasal respiration is held responsible for disturbances in the development of the thorax and abdomen.

The *treatment* of hypertrophy of the pharyngeal tonsil is limited to its operative removal. A large number of instruments have been devised for the purpose, of which the Beckmann modification of the Gottstein curette is perhaps most frequently used. The instrument is inserted behind the palate until it rests against the posterior edge of the septum; the cutting edge of the curette is rotated around an axis corresponding to the size of the epipharynx and the tonsil is readily detached *in toto.* The separated tonsil falls into the lower pharynx and is expelled. In the instances in which the patient " swallows the mass," the maneuver probably has not succeeded. The extent of the bleeding varies widely. Fatal hemorrhage has been found to be associated with *hemophilia.* The wound heals in from eight to fourteen days. Because of the danger of forcing infection into the middle ear, *the pharynx should not be lavaged.* Narcosis is essential; local anesthesia cannot be accomplished in this situation.

Hypertrophy of the Palatine Tonsil.— The *etiology* of hypertrophy of the palantine tonsil is no more clearly understood than is that of the pharyngeal tonsil. The organ is often the seat of inflammatory foci which are regarded as tuberculous in character. There is no doubt that the tonsil is often the port of entrance of general infections and that this occurs more frequently when these organs are the seat of hypertrophic changes.

The enlargement of the tonsil is a familiar spectacle and varies

greatly in extent. The organ protrudes into the arch of the palate, unless it is adherent to its bed between the pillars of the fauces — a common occurrence. Close scrutiny reveals the enlarged tonsil, either smooth or irregularly excavated. The excavations are flecked with yellowish plugs of detritus. In children they are soft, in adults, hard in consistence. The extent to which the tonsil extends downward can be determined only with the mirror. This should always be ascertained, especially as the dependent portion of the organ is frequently responsible for recurrent attacks of acute tonsillitis.

The *clinical picture* of hypertrophical tonsils is exceedingly variable. Not a few of the disturbances ascribed to the palatal tonsils are really due to the presence of a coexisting enlarged pharyngeal tonsil. Simultaneous involvement of these organs is the rule. The chief symptom of enlargement of the palatal tonsils relates to interference with deglutition. Pain is present only during an acute inflammation. However, a feeling of discomfort and cough are likely to be present between the attacks. Very large tonsils interfere with breathing, especially at night, when they cause the patient to snore. The most common and distressing complication of hypertrophy of the tonsils consists in frequent *acute inflammatory exacerbations* of the subacute or chronic process. Regression of the affection may occur after puberty. This, however, is not constant. A certain degree of restitution to the normal at times follows spontaneous or therapeutic evacuation of the tonsillar crypts and the healing of a peritonsillar abscess.

In the *treatment*, tonsillotomy, slitting of the tonsillar crypts and tonsillectomy are taken into consideration.

Tonsillotomy (amputation of the tonsil) consists in removal of the protruding portion of the organ. As the infected crypts extend to the capsule, the operation attains the object only when the amputation includes removal of the intrapalatal portion of the tonsil. In order to accomplish extracapsular removal of the tonsil with the tonsillotome, the organ must be delivered from the fauces, grasped with hooked forceps, drawn toward the median line and sectioned at its base. As the tonsil is often adherent to the pillars of the fauces, it must first be released; this is readily accomplished by a specially constructed hooked knife. Excision of a sector of the fauces increases the danger of secondary bleeding, which is often followed by impairment of the singing voice. At the present time the tonsil is enucleated as follows: The patient is narcotized, the narcosis being maintained by spraying ether vapor into the mouth; a suction apparatus keeps

the operative field clear of blood; the tonsil is grasped and separated from the pillars of the fauces as stated above; a snare (cold) is passed over the forceps and carried around the tonsil and the section made close to the base of the latter; the few bleeding points are clamped for a few minutes, and the same procedure is executed on the opposite side. The pharyngeal tonsil is removed at the same sitting.

Slitting of the tonsillar crypts is a conservative procedure used in cases of moderate hypertrophy and slight general disturbances. The tonsil is delivered into the median line, as stated above; the crypts are opened with a long, slender knife and emptied of detritus. The procedure is not much practiced at this time. Removal of the tonsil with the finger is also rarely performed.

Hemorrhage following tonsillectomy is usually inconsiderable and is almost always controllable in the manner stated. The "bugbear" of injury to the internal carotid may be dismissed with this mention. Uncontrollable bleeding occurs in hemophiliacs, although it has also happened from other causes (diabetes, nephritis, etc.). In these instances, ligature of the external carotid artery is justified.

After the operation the patient is kept in bed for twenty-four hours and is nourished with liquid food. The fibrinous exudate which covers the wound surfaces must not be disturbed.

Lues, Tuberculosis, Lepra, Scleroma, and Glanders of the Pharynx.—Of the inflammatory processes of the pharynx caused by specific excitants, *lues is by far the most frequent.*

The *primary lesion* of lues occurred on the tonsil 504 times in 10,265 cases of extragenital infection collected by Münchheimer.[5] The infection may be conveyed by coitus praeter naturam or by contaminated food, table implements, etc. As the lesion is attended with a severe local reaction and the lymphatic glands are greatly involved, the process is likely to be mistaken for malignant disease, especially when there is much sloughing of tissue in the pharynx. The *diagnosis* is not difficult, if lues is thought of. The spirochete is often present and an injection of salvarsan is followed by healing with astonishing rapidity. The simultaneous presence of *plaques muqueuses,* especially when these show a tendency to ulcerate, is of great diagnostic assistance.

Gummatous lesions are also frequently located in the pharynx and are usually situated on its posterior wall and on the soft palate. The gummata are often multiple, break down early, and in healing cause grave distortions of the tissues. When the lesion is located in the

epipharynx, it is likely to escape notice unless disturbances in deglutition cause the surgeon to make a posterior rhinological examination.

Tuberculosis of the pharynx appears in its lupous form or as an ordinary infiltration and ulceration. The character of lesions of this sort affecting the tonsil has been revealed by the microscope (Seiffert[6]).

The *lupous form* occurs by extension from the lip by means of the mucosa of the mouth and frequently involves the larynx. It presents a picture very similar to the process on the face and, like the latter, shows a tendency to heal. In most cases, unless the larynx is involved, the patient does not suffer much inconvenience. In advanced cases deglutition is accomplished with difficulty. The outcome depends upon the laryngeal and pulmonary involvement. The *treatment* is similar to that employed in efforts to relieve tuberculosis situated elsewhere in the body, i. e., curettement or excision of the local lesion and the use of general sustaining nourishment, etc. The local application of iodoform paste seems to be of benefit.

The *infiltrating form* of pharyngeal tuberculosis soon ulcerates. It most often invades the soft palate, where it presents the typical picture of a mucous membrane lesion of this class. If the ulcer destroys large areas of the palate, it is not easily differentiated from a luetic lesion. Microscopical examination of an extirpated section is likely to remove any doubt in this connection. It must be remembered that pulmonary tuberculosis and a luetic pharyngeal lesion may coexist. The *treatment* is similar to that of the lupous form of the disease.

Lepra of the pharynx probably occurs more frequently than is generally believed. The process is at first nodular in character which soon leads to cicatricial shrinkage. See Part II, chap. xxiv. See also Michelson,[7] Bergengrün.[8]

Scleroma of the pharynx appears as a part of the same process in the nose (Part II, chap. xxvi; Pieniazek[9]).

For *glanders* see Part II, chap. xviii.

Injuries and Foreign Bodies in the Pharynx.— Of the injuries of the pharynx, those caused by burns and chemical cauterization (attempted suicide) are of especial interest because of the distortions and stenosis which follow repair. When the pharynx is incised from without (also frequently in connection with attempted suicide), the development of distressing fistula can only be prevented by exact suture of the divided mucosa and its inversion by means of a second layer of sutures introduced into the pharyngeal wall. The outer wound must be drained in all instances.

Foreign bodies in the pharynx are usually bone fragments, fish bones, and needles, which are introduced with articles of diet and are most frequently located in the sinus pyriformis or the deeper portion of the pharynx. They are located with the mirror or the palpating finger. The Röntgenographic finding is likely to be of great assistance. If the substance is deeply situated, the esophagoscope may have to be used to determine its exact location. Foreign bodies in the sinus pyriformis provoke edema of the aryteno-epiglottic fold, thus endangering the patient's life from strangulation. In these cases the prompt performance of a tracheotomy is a life saving measure. This permits careful exploration of the seat of the obstruction, and when suppuration occurs incision of the inflamed area is very likely to be followed by expulsion of the offending agent. In a certain number of cases the abscess gravitates to the neck and must be opened in this situation. The foreign body may then be expelled through the external wound.

Stenoses and Distortions of the Pharynx.— Most stenoses of the pharynx are the result of a healed luetic lesion, although a few follow chemical cauterization.

Cicatricial coalescence at the junction of the mesopharynx and epipharynx is due to adherence of the soft palate to the wall of the pharynx. Complete atresia of the nasopharynx is rare; usually, a small opening persists. Patients afflicted in this way are mouth breathers and have no sense of smell. In the incomplete form, fluids are likely to flow from the mouth into the nose.

Cicatricial stenosis at the site where the mesopharynx communicates with the mouth and the hypopharynx is always partial, although deglutition is more or less interfered with and the voice is also affected, especially when the base of the tongue is involved in the process. In not a few instances, cicatricial bands are located at the upper end of the larynx.

The *treatment* of this class of cases is not very satisfactory. Even during the stage of healing, it is hardly possible to prevent the formation of the strictures. When cicatrization is completed, efforts at reëstablishment of a physiologically sufficient opening may be made. *Gradual* dilatation is practiced in connection with obstruction at the hypopharynx. It may be followed by severe reaction and is, therefore, usually preceded by tracheotomy. Division of the scar tissue is followed by return of the obstruction, unless this is supplemented by per-

sistent and prolonged use of the bougie. Gastrostomy may be necessary (v. Eicken[10]).

Benign Tumors of the Pharynx.— So-called typical nasopharyngeal polypi possess considerable surgical importance. Histologically they are true fibromata. Despite the fact that at times they break through the surrounding bony structures they never form metastases. The vascularity of these growths varies widely. Some are nourished by vessels the size of the radial artery. However, most of them possess larger or smaller vascular plexuses, some of which present a cavernous form.

FIG. 815.— SAGITTAL SECTION OF A NASOPHARYNGEAL POLYPUS.

The tumors spring from the base of the skull; usually from one of the bony processes. Their area of origin is in the "fibrocartilago basilaris," which is covered with periosteum and extends from the posterior edge of the nasal septum, along both wings of the sphenoid bone and the pterygoid fossa, and then turns downward and backward to include the pharyngobasilar fascia, the cartilages of the eustachian tubes, and the petro-occipital syndesmosis. The tumors are covered with mucous membrane, except when this has been destroyed by trauma and consequent ulceration. The latter frequently gives rise to bleeding, though this is not often menacing.

Obstruction to nasal breathing is the first indication of the presence of the tumor. This is soon followed by a more or less profuse mucous or purulent discharge. The pernicious effect of the growth is the result of its rapid increase in size. At first the tumor grows into the epipharynx and its neighboring nasal cavities on one side and then into those of the other. From the nose it gains access to the sphenoid, maxillary, frontal and ethmoid sinuses, all of which it ultimately fills up, breaking through their walls, and, in not a few instances, extends into

the cranial cavity. From the sphenoid sinuses the process often invades the orbital cavities, though this is more likely to occur in connection with sarcomata. During all of these extensions, the character of the tumor remains benign, and it may be lifted from the respective cavity of origin without difficulty. It is a singular fact that in a certain number of instances the tumor undergoes considerable atrophy after puberty or after incomplete extirpation.

The *clinical findings* are fairly uniform. As a rule the mass fills the entire nasopharynx. Its upper and anterior surface is usually smooth, while the portion obtruding into the pharynx is likely to be ulcerated. Its dimensions may, in a measure, be determined by visual examination and by palpation; frequently it permits of some motility. Its insertion is not easy to determine, so that in making an operative effort at relief one must be content with the empirical assumption that the tumor has sprung from the under surface of the sphenoid bone or the basilar process.

Fig. 816.— Retromaxillary Fibroma.

The *distortions* caused by the growth are likely to be extensive. The nasal process lifts the wing of the nose, the nasal bones are separated and exophthalmos is often marked. Invasion of the maxillary sinus is characterized by protrusions of the cheek, and the temporal fossa is often obliterated. These deformations, together with the open mouth, present a characteristic picture (Fig. 816).

The *differential diagnosis* takes into account the ordinary nasal polypi which protrude into the epipharynx. These are bluish or gray in color, while fibromata are dark red. On palpation, fibromata are hard, polypi soft, in consistence. Bleeding and extension into the contiguous cavities are essentially the privilege of fibromata. However, the latter characteristics are also possessed by sarcomata. Differentiation between sarcoma and fibroma is, unfortunately, not easy.

The fact that a fibroma, even when invaginated in a bony sinus, is somewhat movable, is helpful. A neoplasm protruding from the retropharyngeal tissues must also be taken into account. Chondromata and teratomata in this situation are rare; the former is distinguished by its hard consistence.

The *prognosis,* despite the benign character of the growth, is not good. An unfavorable outcome results from bleeding, invasion of the cranium, and meningitis. Very large tumors interfere with deglutition and respiration.

The *treatment,* in small tumors, consists in their destruction by the galvanocautery. *Extirpation* by the direct route may be accomplished by means of the snare. The manipulation is not easy and requires considerable skill in rhinological technic. *Avulsion* of the growth *in toto* with forceps introduced through the mouth has often been successful; the measure is attended with considerable bleeding. Extensive growths are best attacked by preliminary exposure of the pharynx (p. 1694).

Other benign tumors of the epipharynx and the mesopharynx are similar in character to those described in the general part of this work (see Tumors, Part VI).

Teratomata occasionally spring from the dome of the pharynx. They vary greatly in size and are distinguished by their epidermal covering, which usually possesses some hair (the hairy polypi of Reuter[11]). Extirpation of the tumor through the mouth presents no difficulties.

Pedunculated fibromata occasionally arise from the posterior rim of the choanæ and from the pillars of the fauces, and hang down into the pharynx. They bear considerable resemblance to polypi and are treated as such. So-called *lymphadenoid polypi* are also found in this situation. These are attributed to displaced portions of the tonsillar anlage. Another form of tumor of similar firm, but somewhat coarser, structure found in this region is regarded as a *congenital dermoid.*

Enchondromata and osteomata springing from the basilar process have already been alluded to in connection with nasopharyngeal fibromata.

Of the *tumors composed of vessels* occurring in the mesopharynx *cavernous angiomata* are the most common, although occasionally *lymphangiomata* also make their appearance in this situation. The angiomata do not attract attention unless hemorrhage suddenly de-

velops. In view of the great vascularity of the growths, they are best attacked with the galvanocautery.

Of the benign tumors occurring in the mesopharynx, *papillomata* are the most frequent. These are warty in structure and are covered with an epidermoid layer. Disturbances are caused only when the tumors possess long pedicles and provoke irritation of the air passages (tickling, gagging). They are readily ablated with the scissors.

Of the *cystic tumors,* small retention cysts of the glands of the soft palate are the most frequent.

Mixed tumors of the pharynx occupy a position between benign and malignant growths. They lie between the pillars of the soft palate, and at times send a prolongation through the pharyngeal fascia, which communicates with a similar, larger tumor of the parotid gland (hourglass parotidopalatal tumors). Usually these tumors are at first benign, but they reach gigantic proportions and cause serious pressure disturbances. As they increase in size, pressure ulceration occurs, which is attended with a picture not unlike that of a brokendown gumma. The peculiarity of the growth lies in the fact that it may remain quiescent for years and then suddenly develop all the characteristics of a malignant process which exhibits the histological picture of a round cell sarcoma.

As long as the process remains benign, the tumor may be shelled out of its bed with ease. The smaller growths may be removed through the mouth, the larger ones must be extirpated by the external route (p. 1691).

Extrapharyngeal tumors which encroach upon the pharynx arise from the base of the tongue and the retrovisceral tissues. Of the tumors of the base of the tongue, large cysts are very likely to protrude into the pharynx. They probably spring from rests of the thyroglossal duct. In addition to this, solid tumors, arising from the same source, consist of accessory thyroid glands (struma). *Extravisceral strumata* arise from the upper pole of the thyroid body and encroach upon the pharyngeal lumen. *Retropharyngeal tumors* spring from the connective tissue of the retrovisceral space. These make their appearance in the varied forms of growths arising in connective tissue, mostly, however, as fibromata and enchondromata originating from the periosteum of the vertebral column. Their *extirpation* must be accomplished by the external route.

Sarcomata of the Pharynx.— Excluding typical nasopharyngeal fibromata, sometimes regarded as malignant, sarcomata in this region

are rare. The diagnosis is very uncertain; even the microscope does not furnish the necessary information in this connection. *Lymphosarcoma* is the most common form in which this class of growth is presented. They spring from the follicles of the pharynx, palate, and lingual tonsil. At first the process has the appearance of a simple hypertrophy, which is usually covered with a white, flecked deposit. As the disease progresses, the neighboring mucosa is infiltrated, until the entire epipharynx and mesopharynx are filled by the tumor. The regional lymph nodes are invaded early in the progress of the disease and may be the first evidence of the process. Later, disturbances of deglutition and respiration dominate the picture, and finally the intrapharyngeal growth breaks down. Sarcomata of this variety are not always readily distinguished from lymphomata, pseudoleukemia and leukemia.

The other forms of sarcoma develop in the tonsil and usually are of the round cell variety. A number of small sarcomatous tumors often invade the pharynx from the upper portion of the larynx, and prevertebral sarcomata also encroach upon the pharynx (Bergeat[12]). Clinically, the former class of sarcomata cannot be differentiated from benign fibromata. They may be extirpated from the mouth. Sarcomata of the tonsil may be extirpated by the external route (p. 1679). The larger lymphosarcomata are not susceptible to operative measures of relief.

Carcinoma of the Epipharynx, Mesopharynx, and Hypopharynx.—Carcinoma most often invades the pharynx from the tonsil. The process is usually rapid, extending first along the retromaxillary lymph nodes and then into those situated along the carotid sheath. The glandular involvement often attracts attention before the carcinoma in the tonsil or pharynx is recognized. The growth is usually of the glandular type, rarely of the squamous. The latter occurs in the hypopharynx in connection with carcinoma of the larynx.

The clinical course and symptomatology of pharyngeal carcinoma varies with its location.

Nasopharyngeal carcinoma presents a picture similar to that of sarcoma in this situation (see above).

Oropharyngeal carcinoma is usually heralded by painful deglutition. This is soon followed by swelling of the cervical glands, which prompts the surgeon to examine the throat, thus uncovering the process in the pharynx. Occasionally, the pharyngeal (or tonsillar) growth is not ulcerated, simply presenting a firm, globular swelling (nodular stage

of carcinoma) which seems to force the tonsil into the pharynx. At this time the process is very likely to be mistaken for a gumma. When ulceration is established, the excavation always has a hard edge (see Carcinoma of Mucous Membranes, p. 1273).

The lesion must be differentiated from tuberculosis (including lupus), actinomycosis, lues, and simple mucous ulcer. The determining factors are taken up in discussing carcinomata of the tongue (p. 1675).

Carcinoma of the hypopharynx is usually a part of laryngeal carcinoma. However, occasionally the disease appears primarily in this situation in the form of an annular zone of thickening, which soon ulcerates and shows a tendency to obstruct the lumen of the lower pharynx. Extension to the sinus pyriformis, the arcus palatopharyngeus and the tongue is the rule.

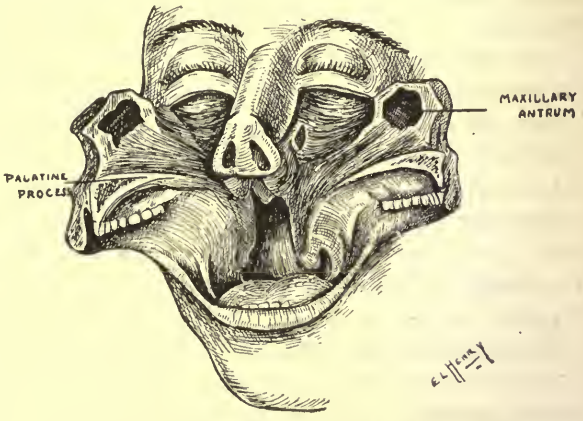

FIG. 817.— EXPOSURE OF THE NASOPHARYNX (v. Langenbeck).

Pain and difficulty in deglutition are the *chief symptoms* of carcinoma of the hypopharynx. This leads to examination and recognition of the lesion. The esophageal sound must be used with care. Direct examination of the hypopharynx with the esophagoscope will lead to a prompt *diagnosis*.

Operative Exposure of the Pharynx.— The principle of all operative measures directed toward removal of growths of the pharynx involves preliminary wide exposure of this part. This applies to benign as well as malignant processes.

Temporary resection of the alveolar processes, together with the hard palate, has already been taken up (p. 1679). The operation gives ample access to the posterior nares, but not to the epipharynx.

Osteoplastic resection of the upper jaw gives a wide exposure of the nasopharynx. The technic of the procedure is similar to that practiced in connection with incision of the superior maxilla (p. 1646),

except that it is carried out on both sides and the cheek flaps are not separated from the bone. The bone and soft parts of both halves of the upper jaw are turned back on each side of the face (Fig. 817), and the nose, after division of the nasal bones and septum, is turned upward. Preliminary ligature of both external carotid arteries and tracheotomy are generally practiced.

The *removal of tumors from the mesopharynx and the tonsil* is best accomplished by lateral section of the inferior maxilla in the manner described in connection with carcinoma of the tongue. While carci-

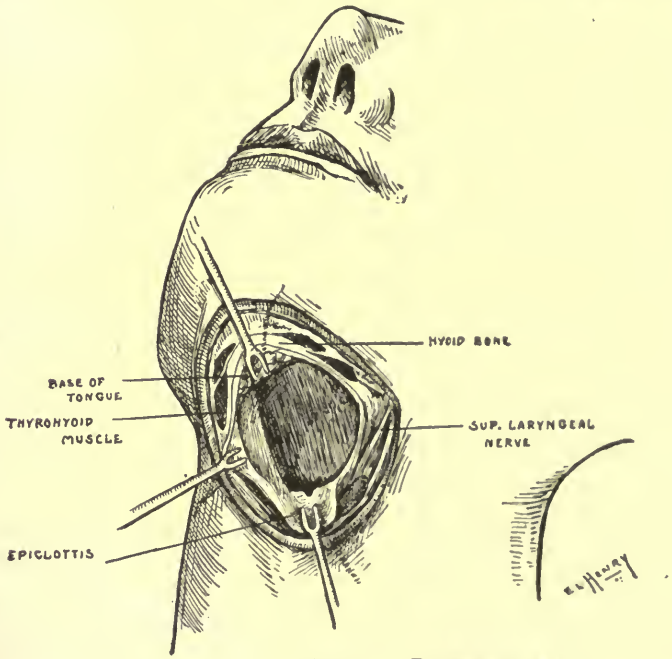

FIG. 818.— TRANSVERSE SUBHYOID PHARYNGOTOMY.

noma of the tonsil has been removed through the mouth, this is not generally regarded as sufficiently thorough to give reasonable security against return of the disease. Of course, the lymph nodes in the neck must be removed at the same time (Krönlein,[13] Citelli,[14] Krause-Heymann[15]).

Of the operations employed for the purpose of *gaining access to the hypopharynx, transverse subhyoid or lateral pharyngotomy* gives ample space.

Tumors of the epiglottis are easily enucleated by *transverse sub-*

hyoid pnaryngotomy. The incision is made immediately over the hyoid bone and extends from one of its ends to the other. The hyoid artery and vein are tied and, after the bone is exposed, the insertions of the sternohyoid, omohyoid and thyrohyoid muscles are divided. The superior laryngeal nerve is avoided. Section of the thyrohyoid membrane exposes the upper portion of the hypopharynx (Fig. 818).

For removal of tumors in the lower hypopharynx, *transverse, combined with lateral, subhyoid pharyngotomy* becomes necessary. Lateral pharyngotomy may be practiced alone. See Bryant.[16] The incision begins in the middle of the hyoid bone, and extends along this bone to the anterior border of the sternomastoid muscle, where it curves upward opposite to the angle of the jaw. The sternohyoid, omohyoid, and thyrohyoid muscles are separated from the lower edge of the bone. The submaxillary glands and the lymph nodes are removed and the thyrohyoid membrane and the mucosa are opened in a transverse direction. When the hyoid bone is lifted with a blunt hook, the entire hypopharynx and its mergence into the esophagus is laid bare.

In order to preserve normal deglutition, the connection between the larynx and the hyoid bone should not be abolished. This is less disturbed by lateral than by subhyoid pharyngotomy.

When the glands of the retromaxillary and submaxillary regions are involved and section of the lower jaw is necessary, this may be readily accomplished by the method discussed in connection with operations for carcinoma of the tongue (p. 1677).

For the relief of *extensive laryngopharyngeal carcinoma* the retrothyroid or retrolaryngeal methods of Quénu-Sebileau[17] or of Rasumowski[18] may be employed. An incision is made along the anterior border of the sternomastoid, and the pharynx invaded by careful dissection of the muscles, vessels and nerves. The technic of the procedure is largely a question of the surgeon's knowledge of anatomy, which it is impossible to supplement here (see Krause-Heymann[15]). Gluck's[19] simultaneous excision of the larynx and pharynx is discussed elsewhere (p. 1754).

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PART XII

SURGERY OF THE NECK

CHAPTER I

MALFORMATIONS OF THE NECK

Congenital Fistulæ and Cysts of the Neck.— Congenital fistulæ are divided into *median* and *lateral.*

The *median fistula* is the result of an eversion of that portion of the epithelial layer of the floor of the mouth which is concerned in the formation of the middle lobe of the thyroid gland. In fetal life, as the thyroid body sinks downward, the canal leading from this gland to the root of the tongue (foramen cecum) is not obliterated. This canal, or duct (thyroglossal duct), runs from the foramen cecum to the hyoid bone. The fistula is primarily incomplete, reaching to the skin of the neck, which, however, it usually perforates later in life. This is in accord with the clinical findings in this connection. The opening lies in the median line of the neck at various levels between the hyoid bone and the jugulum. The canal itself is readily palpated — a hard cord reaching to the hyoid bone. The *treatment* consists in extirpation of the fistulous tract. This may be accomplished under local anesthesia. If portions of the duct are left behind the fistula reappears.

Sistrunk[1] presents the technic in detail. His presentation is excellently illustrated.

In lateral fistula of the neck (fistula of the thymopharyngeal duct) the opening lies between the median edge of the sternomastoid muscle and the median line, between the hyoid bone and the jugulum, either close to the sternoclavicular articulation over the thyroid cartilage or near the body of the hyoid bone. The fistulous tract perforates the subcutaneous tissues, the platysma, and the superficial fascia, and runs upon the surface of the deep fascia parallel to the sternomastoid muscle, crosses the sternohyoid and thyroid, passes between the internal and external carotid arteries, dips under the digastric and ends in the lateral wall of the pharynx (Hildebrand[2]). It is closely associated with the sheaths of the deep muscles. The fistula may be *complete* or *incomplete,* the patent opening being either within the

pharynx or external on the neck, as stated above. The incomplete form may become complete as the outcome of subsequent rupture. The structure of the tract varies and may resemble a tubular gland. Although several fistulæ may be present, the existence of a single tract is the rule. The external opening is small and discharges a mucous or mucopurulent fluid. Temporary closure of the opening is followed by swelling and pain, the latter being increased during deglutition. In internal incomplete fistula, food collects in the tract and gives rise to similar disturbances.

The *diagnosis* may be made certain by the injection of colored fluid and the use of the probe. The latter does not enter the pharynx, but is arrested at the larynx or hyoid bone where the tract bends sharply inward.

The *treatment* consists in complete extirpation of the tract. The intimate relationship that the canal bears to important structures in the neck makes this a difficult problem (Alivisatos[3]).

The *origin of lateral fistulæ of the neck* has been made the subject of considerable investigation. According to Weglowski[4], the fistulous tract emanates from persisting rests of the *thymopharyngeal duct,* which is analogous to the thyroglossal.

Congenital cysts of the neck (serous, mucous, and dermoid cysts) appear in various parts of the anterior portion of the neck. Like fistulæ, they may be divided into median and lateral. The *lateral cysts* usually lie between the larynx and the sternomastoid muscle, but they appear also between the mastoid process and the hyoid bone, and are found occasionally in the lower triangle of the neck. In either event, they may extend over the greater part of the neck. The *median cysts* are usually located above or below the hyoid bone, but are also found between the thyroid cartilage and the jugulum. They are rarely larger than a hen's egg and are not unlike the latter in form. While the superimposed skin is normal in appearance and is freely movable over the tumor, the cyst itself is adherent to the deeper structures. In a sense the cysts are not congenital and do not, as a rule, make their appearance until about puberty. According to the most recent views, *median cysts* take their origin from the *thyroglossal duct,* and the *lateral cysts* from the *thymopharyngeal duct* and correspond in structure to that of the walls of the fistulæ described above (Weglowski[4]). The contents of the cyst is the product of the lining epithelium and is usually either serous or mucous in character; but it may consist of a thick, fatty paste, often mixed with hair. As these

different constituents exist in varying proportion, a classification of the character of the cyst, based on its contents, is not justified. The terms *hydrocele colli, deep atheroma,* and *dermoid cyst* therefore are misapplied.

The topographical location of congenital cysts of the neck corresponds to that of the fistulæ (see above). Lateral cysts are intimately associated with the sheaths of the deep vessels and are often attached to the styloid process and the cornua of the hyoid bone. The median cysts are located above or below the hyoid bone. All cysts of this class grow slowly and do not cause disturbances unless infection supervenes. Their extirpation is beset with considerable difficulty; however, no other treatment accomplishes the purpose (see Chevassu,[5] Cignozzi,[6] Morton,[7] Walther[8]).

Branchiogenic Outgrowths of the Skin of the Neck.— Small outgrowths occasionally appear upon the skin of the anterior portion of the neck; these may consist entirely of skin, or they may contain a certain amount of cartilage. They are located on one, or both, sides, at about midway between the sternum and the hyoid bone; they vary greatly in size (wart, nipple, mushroom), and are likely to be pendulous. The growths take their origin from the second branchial arch and are continuous with the fascia investing the sternomastoid muscle. Most observers regard the anomalies as supernumerary ears, a view which accords with their origin. Their *extirpation* is readily accomplished.

Cervical Ribs.— In 1907 Keen[9] collected forty-three cases of cervical rib. The condition is really that of a supernumerary rib, usually connected with the seventh cervical vertebra. The degree of development varies; the rib may extend only as far as the transverse process, in some instances it merges with the first thoracic rib; in others it reaches as far forward as the costal cartilage and may be attached to the sternum. A cervical rib may be present on both sides, and occasionally two are found on the same side.

The surgical importance of the cervical rib lies in the relationship it bears to the subclavian artery and the brachial plexus. The ARTERY ALWAYS LIES ABOVE THE CERVICAL RIB or, when the latter is too short, in front, never beneath it. In the same way the plexus, which lies beneath the rib, is also subjected to pressure. The scalenus anticus is often attached to the anterior end of the rib, and an intercostal muscle is present between the latter and the first thoracic rib.

The cervical rib of itself does not give rise to symptoms. The

disturbances are due to changes in the circulation and nerve supply. The severity of the symptoms, due to *pressure on the subclavian artery,* varies greatly. In some instances only lessening of the radial pulse is caused, in others, a thrombus extends from the subclavian artery to the periphery. Occasionally, an aneurism of the vessel develops. *Clinically,* the anomaly is attended with reduction in the temperature and pulse of the arm and hand. Gangrene has occurred.

The *symptoms* arising in connection with the *brachial plexus* are those of disturbances of sensation, radiating neuralgic pain, paresthesia, tingling, numbness, and, after a time, the limb feels heavy and weak. While distinct motor paralyses do not occur, the electrical excitability of the nerves is reduced. In a few instances atrophy of the muscles of the hand has occurred. Local trauma, such as a sudden exertion, may precipitate symptoms which attract attention to the condition. Reflex disturbances are analyzed by Hunt.[10]

The *diagnosis* is based on the effacement of the normal supraclavicular depression, which presents a marked protrusion and exhibits a distinct pulsation. Palpation reveals that the protrusion is due to a hard bony mass and that the pulsation corresponds to the subclavian artery. When the rib is very short, neither of these signs is present. The Röntgenogram makes the diagnosis certain.

The *treatment* consists in excision of the supernumerary rib. This is accomplished through the incision employed for ligature of the subclavian artery (p. 1023). The pleura often extends to the level of the rib; its injury is not attended with serious consequences (Taylor,[11] Murphy[12]).

Wry Neck is taken up in Part VIII, chap. iv.

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CHAPTER II

INJURIES OF THE NECK

Burns.— Of the injuries of the neck, burns are of importance because of the great distortions sequential to healing. These should be obviated as far as is possible by suitable posture of the head during repair.

Fractures of the Hyoid Bone.— Because of the mobility and deep location of the hyoid bone, it is not often fractured. *Direct fractures* occur in connection with hanging, the *indirect* by strangulation. The fragments are usually widely separated. The *symptoms* consist in painful deglutition, difficulty in speaking and cough. The fracture is easily palpated. *Reposition* is accomplished by manipulation. *Fixation* is maintained by means of a gypsum or celluloid collar (Noble[1]).

Injuries of the Arteries of the Neck.— Bleeding is an important factor in connection with stab or incised wounds of the neck, especially when the latter are self inflicted. It may be divided into external, internal, and interstitial. In large wounds the blood is usually discharged externally; when a hollow space, such as the trachea or pleura, is opened, a portion of the blood is emptied into it. Extensive infiltration of blood into the tissues occurs when the external wound is small (deep stab wounds involving large vessels). The character of the wound and its location and extent determine the degree of bleeding, which is primary or secondary. Primary bleeding is greatest when a vessel is divided transversly and is always a serious problem in these cases. *Secondary bleeding* occurs after five days (between the fifth and tenth day). It is the result of dislodgment of a thrombus or of infectious destruction of the walls of a vessel or vessels.

It is important to recognize the source of the bleeding. This, in many instances, is difficult, especially if the patient is in shock, when the character of the hemorrhage is very misleading. In most cases it is best to enlarge the wound and carefully explore its surfaces. The outcome of injuries of this sort is largely dependent upon the promptness with which relief is afforded. The wound is temporarily tamponed, its surroundings cleansed, and the surgeon freely exposes the

injury and proceeds systematically to clamp and deligate all the bleeding points. When the injured area is heavily infiltrated with blood it is best to deligate the afferent artery in its continuity and then explore the site of the injury as stated. In injuries of the common carotid and subclavian arteries, repair of the walls of the vessel by suture (see Arteriorrhaphy, Part V, chap. v) may be undertaken, if this is warranted by the general condition of the patient.

Injuries of the Veins of the Neck.— Although asepsis has greatly lessened the occurrence of infection, injuries of veins in the neck still present a certain menace in this connection. To this may be added the dangers of fatal bleeding and the aspiration of air. As the veins in this situation do not possess valves and are in direct communication with the cranial sinuses, fatal cerebral anemia may occur when the latter are suddenly emptied of blood.

Aspiration of air is most likely to be associated with injuries of veins situated at the root of the neck and those intimately associated with the fasciae and muscles, which prevent their collapse when divided. The entrance of air into the vein is attended with a bubbling or gurgling sound and in a few minutes the symptoms of interference with the circulation appear. These are by no means always followed by a fatal outcome (see Air Embolism, p. 586).

Immediate double ligature of a divided vein should be practiced. Tamponade of the wound is unreliable and is justifiable only when the injury in the vein is inaccessible. In lateral or incomplete transverse section of large veins (internal jugular, subclavian, innominate) repair by suture may be made. The measure succeeds in a large number of cases, provided the wound is not infected (Tiegel[2]).

Injuries to the Thoracic Duct.— The deep situation of the thoracic duct in the neck protects it from trauma, so that its isolated injury, except during the employment of operative measures executed in the subclavian triangle, is rare. In this connection it must be remembered that the entrance of the duct into the angle formed by the internal jugular and subclavian veins is subject to variations, and also that the duct may be double.

The *danger of the injury* lies in the permanent discharge of chyle upon the surface of the body which deprives the patient of important elements concerned in metabolism. However, in most instances, tamponade of the wound and repair of the duct by suture, when possible, is followed by reëstablishment of the flow of chyle into its normal channel (Schwinn,[3] Fredet,[4] Parsons and Sargent[5]).

Injuries of the Nerves of the Neck.— Division of the vagus, when limited to one side, is followed by temporary increase in the pulse rate, which is not dangerous to life. The subsequent development of pneumonitis is not ascribable to section of the nerve (Jordan-Völcker[6]). *Paralysis of the recurrent laryngeal nerve* is a constant result of division of the vagus, and impairment of deglutition is a frequent outcome of section of the motor fibers.

Manipulation and traumatic irritation of the nerve is far more serious in its consequences than is division. In a case operated upon by the writer the accidental inclusion of the vagus in the grasp of an artery forceps was promptly followed by death. In extensive operations involving manipulations of the nerve its infiltration with a 1 per cent novocain solution is permissible.

Injuries of the cervical sympathetic occur in connection with extirpation of large tumors in this situation. Irritation of the nerve is attended with dilatation of the pupil, widening of the slit of the eyelids, protrusion of the bulb, and coldness of the side of the face. Section is followed by myosis, ptosis (from paralysis of the smooth fibers of the muscles of the eyelids), redness and heat of the side of the face.

Injuries of the brachial plexus are most often produced by projectiles or splintered fractures of the clavicle, either of which is usually accompanied by simultaneous injury to important contiguous vessels. The *symptoms,* consisting of sensory and motor paralyses, vary with the extent of the nerve fibers involved. The question of primary or secondary repair of the plexus is decided by the condition of the wound.

In injury of the spinal accessory nerve, involvement of its external branch is of surgical importance. This frequently occurs in connection with operations in this region. The lesion is followed by incomplete paralysis of the sternomastoid and trapezius muscles, both of which are also supplied by branches of the second and fourth cervical nerves.

Aneurisms of the Neck.— As in other portions of the body, aneurisms of the neck are divided into true and false. Of the problems presented in the general part of this book (Part V, chap. v) only those possessed of especial local interest will be taken up here.

Aneurism of the common carotid artery is attended with special *pressure symptoms,* dyspnea, difficulty in swallowing, neuralgia, and paralysis of the hypoglossal, recurrent laryngeal and sympathetic nerves. Headache, vertigo, insomnia, and temporary hemiplegia are attributed to cerebral anemia.

In making the *diagnosis,* lymphoma of the intercarotid gland must be excluded.

Of the various methods of *treatment,* double ligature of the sac or *aneurismorrhaphy* give the best results (Delore[7]).

Aneurism of the external carotid artery is usually treated by ligature of the common carotid, although double ligature of the sac and its extirpation is also practiced with considerable success.

Aneurism of the internal carotid artery (extracranial portion) is most often the result of necrosis and perforation of the walls of the vessel, the outcome of deep seated inflammatory processes on the neck. Pressure symptoms (dysphagia) and severe headache occur very early in the course of the development of the lesion.

Central ligature of the artery itself is the *treatment* of choice. If this is not feasible, ligature of the common carotid is performed. Aneurismorrhaphy has been practiced by McMullen and Stanton[8].

Aneurism of the innominate artery is not always easily diagnosticated. A conclusion in this connection should not be hastily arrived at. Of the operative methods of relief, *peripheral* ligature — i. e., of the common carotid and subclavian arteries — is the method of choice. With the view of obviating the lodgment of a liberated embolus in the brain, the carotid should be tied first (Guinard[9]).

In aneurism of the third portion of the subclavian double ligature and extirpation of the sac may be practiced. Aneurismorrhaphy has been executed with success (Savariaud[10]).

Arteriovenous aneurism of the *common carotid* and *internal jugular* is of especial interest on the ground that, in a number of instances, immediate exposure of the injury and repair by suture of the opening in the vein and artery is frequently followed by restitution to the normal (Herzen[11]. See also Sencert[12]).

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CHAPTER III

DISEASES OF THE NECK

Acute Phlegmons and Abscesses.— Acute phlegmons and abscesses of the cellular tissues of the neck are caused by (a) direct infections of wounds of the skin and mucosa; (b) extension of infection from neighboring inflammatory processes; (c) conveyance by way of the lymph channels; (d) hematogenous invasion.

The peculiarities of these processes are due to the anatomical conditions presented in the neck.

Submaxillary phlegmons and abscesses are by far the most frequent; they are usually lymphogenous in origin and arise in connection with carious teeth, periostitis of the mastoid process, or ulceration of the gums; or from lesions of the face. As a rule, the inflammation takes on the form of a periadenitis and extends from the anterior edge of the sternomastoid muscle to the chin. This region is markedly swollen and infiltrated and presents the typical picture of inflammation. At times, the hardness of the infiltrate justifies the use of the term *wooden phlegmon* (p. 325). In a certain number of cases, the invasion of the deep cellular tissues of the neck is very extensive, presenting a condition of affairs (often fatal) to which the term *Ludwig's angina* (p. 325) has been applied. The process is at this time regarded as a particularly virulent form of infection of the tissues mentioned. The contiguous cellular tissue of the floor of the mouth, larynx, and pharynx are infiltrated by inflammatory edema (Price,[1] Turner,[2] Thomas[3]).

The *symptoms* are fairly uniform. Aside from those due to systemic disturbances, dysphagia and swelling appear promptly. The head is held inclined toward the affected side, and the swelling of the floor of the mouth causes the tongue to protrude from the mouth, which can only be slightly opened. Mastication is impossible; the saliva dribbles, and the breath emits an offensive odor. Death occurs from exhaustion or may be the outcome of edema of the glottis. In less severe cases, the process goes on to the formation of an abscess which ruptures into the mouth or upon the surface of the neck.

The *treatment* consists in free incision of the infiltrated tissues; fluctuation should not be awaited. The sooner the pressure on the tissues is relieved the better. *Tracheotomy* may be necessary.

Submental phlegmons usually arise in connection with infection of the submental lymph nodes derived from rhagades, ulcers of the frenulum, furuncles of the chin, etc. They show little tendency to extend. The incision should be vertical.

Phlegmons and abscesses of the connective tissue of the vessel sheaths occur as a part of angina. The ports of entrance of the infection are carious teeth, the scalp, otitis, ozena, and stomatitis. The affection often develops during scarlatina. The *clinical picture* is very similar to that described in connection with submaxillary phlegmon, except that the swelling and infiltration appear at first along the sternomastoid muscle, from which it extends in all directions, especially downward to the supraclavicular region. In some instances, the process gains access to the anterior mediastinum, the pharynx, the esophagus or the trachea. *Early incision* is indicated. Search for exudate in the deeper tissues is made with a closed forcipressure.

Abscesses of the supraclavicular region originate in the deep cervical glands or gravitate from the upper areas of the neck. The exudate usually perforates the platysma, but in some instances it proceeds along the sheath of the vessels and gains access to the axilla.

Diffuse phlegmon of the neck often presents itself in a particularly virulent form, especially in children (during acute infectious diseases), and in adults afflicted with phthisis, diabetes, malignant tumors, alcoholism, etc. Phlegmon of the carotid sheath (*absces profond de Chassignac; phlegmon large du cou de Dupuytren*) is particularly menacing. Many of these cases die from the severity of the infection, while a certain number progress to abscess formation. This form of phlegmon is very likely to perforate into one of the large vessels of the neck (Jordan-Völcker[4]).

Acute lymphangitis of the neck usually attends one of the processes just discussed.

Chronic Inflammatory Processes of the Neck.— In the development of chronic inflammatory processes of the neck, tuberculosis, actinomycosis and syphilis play the leading rôles. Of these, tuberculosis is the chief aggressor.

Chronic lymphadenitis (simple hyperplastic lymphoma) is a term applied to enlargement of the lymph nodes caused by the prolonged absorption of irritating substances of a chemical or bacterial nature;

the nodes do not show a tendency to suppurate (see Diseases of the Lymphatics, Part V, chap. iv). The provocative factor consists in one of a variety of conditions, such as eczema, catarrh of the mucous membranes of the nose and pharynx, chronic blepharitis, hypertrophy of the tonsils, ulceration of the gums and *caries of the teeth* (Stark[5]). The affection is especially prevalent in children.

The *differential diagnosis* takes into account tuberculosis and malignant disease; only the former may be recognized by the microscope. The *treatment* consists in removal of the cause.

Tuberculosis of the lymph nodes of the neck (tuberculous lymphoma) is very common. An elaborate regional classification is offered by Jordan and Völcker.[4] The process appears in the several forms.

1. *In the purely hyperplastic form*, the bacillus does not provoke nodular thickening of the glands. This is simply a very mild form of the infection.

2. *In hyperplasia, with the formation of nodules*, the nodes are visible to the eye and vary in size from that of a hazel nut to a hen's egg, depending upon the severity of the infection.

3. *Caseation* occurs when the tuberculous tissue breaks down; this may develop in only a part of the infected nodes. *Liquefaction* is an advanced stage of caseation.

4. *Fistula formation* is the outcome of invasion of the perinodal tissues by extension through the capsule of the gland. The latter undergoes a hyperplastic thickening, in which it becomes adherent to the contiguous nodes and to the surrounding tissues. Finally, this connective tissue growth also breaks down and the process invades the skin, where small abscesses are formed which empty their contents on the surface of the body. This presents conditions exceedingly favorable to the entrance of pyogenic excitants, in which event a so-called mixed infection develops. On the other hand, the expulsion of disintegrated tissue is not rarely followed by spontaneous healing of the process.

The *etiology* of tuberculous lymphadenitis of the neck is chiefly concerned in the manner in which the infection invades the lymph channels. Primary lymphatic tuberculosis probably does not exist and its hematogenous origin is regarded as unlikely. The theory that the process extends upward from the lung is equally improbable. There is no doubt that the *chief ports of entrance in tuberculous lymphoma of the neck are located in the upper portions of the head and neck;* i. e., the process is to be regarded as secondary and is due

to the migration of the bacilli by way of the lymph channels. It seems to have been shown that the excitants may penetrate through intact skin and mucosa; there is no doubt that they may enter through a patch of eczema (Fraenkel[6]). The usual ports of entrance are carious teeth (Stark[5]), the mucosa of the gums (Mathews[7]), and the tonsils, including the pharyngeal tonsil (Judd,[8] Eisendraht,[9] Osborne[10]). The affliction is rare in well nourished individuals and is most common in children.

The *clinical picture* is that of a slowly developing enlargement of the lymph nodes of the neck attended with moderate or no constitutional disturbances. The nodes are, at first, movable and may remain so. When periadenitis occurs they adhere to the surrounding tissues. In some instances the glandular enlargement is very great, the process presenting a large nodular tumor. Before rupture, tuberculous abscesses appear in the form of a low grade of inflammation involving the superficial tissues.

The *diagnosis,* in cases in which abscess and fistulae are formed, is not difficult; however, this stage bears considerable resemblance to actinomycosis, and perhaps to gumma. In elderly persons, lymphosarcoma must be excluded with the aid of the microscope. Difficulties arise when there is no periadenitis. In children, confusion with simple chronic hyperplasia is common. In doubtful cases the microscope must decide the question. In the second and third decennium, pseudoleukemia must be excluded; clinical differentiation is impossible. Malignant disease of the lymph nodes must also be taken into account.

In the *treatment,* prevention of invasion of the lymph channels must be considered. Lesions of the upper neck, the throat and the head must receive proper care. The constitutional treatment of tuberculosis is exceedingly important. The use of the Röntgen ray seems to possess considerable value (Pirie,[11] Kienböck,[12] Holding.[13] See also Part II, chap. xxiii).

The *operative treatment* is indicated when the capsules of the glands are involved. It should be carried out before periadenitis is advanced. When the nodes are movable, the technic is not difficult; later in the process the anatomical landmarks are likely to be effaced. The incision should be placed so that subsequent scarring is reduced to a minimum. For invasion of the *upper triangle of the neck,* the *incision* extends from the tip of the mastoid process to the middle of the hyoid bone, passing one finger's breadth below the angle of the jaw. The digastric, stylohyoid, geniohyoid, mylohyoid and hyoglossus muscles lie

above; the sternohyoid, thyrohyoid and omohyoid below, and the sternomastoid behind the line of exposure, which also permits of avoidance of the vagus, sympatheticus, accessorius, the lower branch of the facial, the hypoglossal, lingual and glossopharyngeal nerves. The lower triangle is readily invaded through an incision midway between the one just described and the clavicle and parallel to the former. Enucleation of non-adherent glands through an incision located at the back of the neck (Dollinger[13]) is a dangerous measure. When periadenitis is present, the line of incision is decided by the extent of the process. In these cases, the curet must often be resorted to. In extensive involvement, extirpation of the process is accomplished through wide, atypical incisions, which include the formation of flaps and also provide for division of the sternomastoid muscle in order to gain access to the infected nodes (Fig. 819) (de Quervain,[15] Plummer,[16] Parker,[17] Courtney[18]).

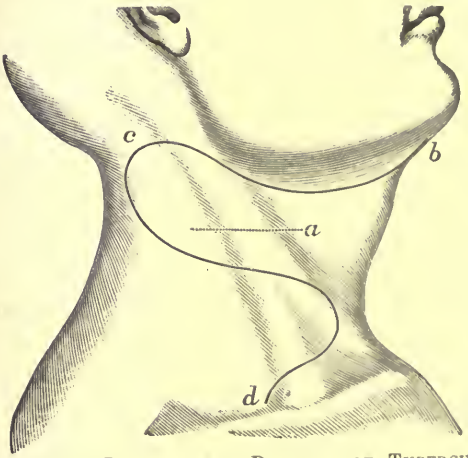

FIG. 819.—INCISION FOR REMOVAL OF TUBERCULOUS LYMPH NODES OF NECK.

a, Point of division of sternomastoid muscle; *b*, *c*, *d*, line of incision (Hartley).

The *end results of operative treatment* seem to justify its employment. In this connection the statistical review of Dowd[19] is particularly illuminating. Of 245 cases operated upon by himself, only 45 required secondary operation.

Lues of the Neck.—*Primary luetic adenitis* occurs in the neck in connection with chancre of the lip, the tongue and the tonsil. The process is attended with pain and swelling and must be differentiated from acute suppurative adenitis.

In the *secondary stage* of lues the cervical glands are almost always involved and often remain more or less enlarged for years. The nodes do not reach a great size, are painless, and are freely movable; i. e., periadenitis does not develop, an important differential diagnostic point as regards tuberculosis.

Tertiary luetic adenitis in the neck appears in the form of a gum-

matous lymphoma. The lesion is rare and presents itself at first in the form of hyperplasia of a single nodule, or group of nodes, which soon coalesces with the surrounding tissues (periadenitis luetica), becomes adherent to the skin and ultimately breaks down; this is followed by the formation of a typical luetic ulcer. In the *diagnosis,* the therapeutic test should not be omitted; however, as stated in connection with carcinoma in general (p. 1283), this must not be extended over a period of more than two weeks.

The *sternomastoid muscle* is at times the seat of a peculiar luetic infiltration (luetic myositis) which may be diffuse or circumscribed (Honsell[20]). Elliot[21] has collected a number of cases of *lues of the hyoid bone.*

Actinomycosis of the Neck.— Excluding the jaw and the cheek, the neck is most often invaded by the ray fungus. As a rule, the infection in this situation is a part of that developing in the mouth, where the excitants gain access to the tissues through carious teeth, ulcers in the mucosa, and through the seemingly ever receptive tonsil. The lesion usually makes its appearance in the region of the lower jaw and extends downward by direct extension — not by the lymphatics. The process presents the *clinical picture* of a slowly progressing low grade of destructive inflammation which soon develops numerous fistulous tracts. The exudate contains the ray fungus. A peculiarity of the disease is that it heals in one area, while extending to another. The *diagnosis* is certain only when the microscope reveals the causative fungi.

The *treatment* consists in curettement of the lesion and the administration of *iodin.* The latter does not have any direct effect upon the ray fungus, but seems to place the tissues in a condition to resist its propagation. Rosenfeld[22] reports favorable results following the injection of Lugol's solution (3 per cent), which he combines with the administration of potassium iodid in increasing doses (see also Shiota[23] and Part II, chap. xix, of this work).

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CHAPTER IV

TUMORS OF THE NECK

Cystic Tumors.— Congenital cystic hygromata (cystic lymphangiomata) occur early in life. They usually begin in the subcutaneous fatty tissue and extend into the deeper connective tissue, though at times a hygroma takes its origin from the sheaths of the large vessels. As the growth enlarges it extends between the various parts of the neck (esophagus, trachea, etc.) and gains access to the mediastinum, the pharynx and the mouth. It does not usually possess a connective tissue capsule, a circumstance which much enhances the difficulty of its removal. The benign character of the process is evinced by the fact that it does not destroy the tissues contiguous to it.

Clinically, a hygroma presents itself in the form of a laxly fluctuating tumor with a smooth surface. It is translucent through the atrophic superimposed skin. The latter is occasionally thickened (elephantiasis) and the superficial veins are dilated. Pressure does not reduce the size of the tumor. The growth is composed of dilated lymph vessels (Wegner[1]) and its contents consist of a clear serous or milky fluid which, when mixed with blood, may be brown in color; it readily coagulates. The interior of the cyst presents a number of cavities of varying sizes. The tumor grows very slowly, but often assumes enormous dimensions. In the *diagnosis,* branchiogenic cysts must be taken into account; these are recognized by their typical location. In doubtful cases aspiration clears up the situation. The *treatment* consists in extirpation of the cyst — not an easy problem (Pätzold[2]).

Blood cysts in the neck owe their origin to fetal arrest of development; a preëxisting branchiogenic cyst; dilatation of a vein; a cavernous angioma, or a lymphangioma. Their *recognition* is based on the facts that the cyst enlarges during coughing or other exertion and that it may be emptied by pressure and does not pulsate. Extirpation of the cyst is the *treatment* of choice; however, obliteration of the sac has been accomplished by prolonged pressure with bandages and by the injection of iodin; the latter is not without danger.

Atheromata of the skin of the neck are not common (see Part VII, chap. i).

Bursal cysts of the hyothyroid region develop in connection with mechanical irritation and rheumatism. Enlargement of the bursae over the thyroid isthmus may be mistaken for a cyst of the latter. On the other hand, a positive diagnosis of a cystic bursa in the thyrohyoid region is not feasible until the lesion is exposed.

Echinococcus cysts of the neck are exceedingly rare. The *diagnosis* is made only during extirpation of the growth (Bevers[3]).

Blood Vessel Tumors of the Neck.— Angioma simplex appears in the neck in the form of a flat nevus and subcutaneous angiomata. The latter develop most often in the region of embryonic clefts (fissural angiomata). They may be destroyed by ignipuncture or excised.

FIG. 820.— HYGROMA OF THE NECK.

Angiomata cavernosa appear in the superficial and deep forms. As a rule, they develop in adult life and are often the outcome of the simple form of angioma. Like all tumors of this sort, they may be emptied by pressure and return when this is released. While ignipuncture and the injection of alcohol has been followed by obliteration of the vessels, extirpation of the process is desirable. This is, however, attended with some difficulties (Eisenreiter[4]).

Solid Tumors of the Neck.— Fibromata of the neck are divided into the superficial and deep. *Superficial fibromata* arise from the cellular tissues of the skin and from the subcutaneous tissues (see v. Reckling-

hausen's Disease, Part V, chap. i). *Deep fibromata* originate from the aponeuroses and the periosteum of the vertebrae (Jordan[5]), and may reach the size of a child's head.

Fibromata grow slowly and cause disturbances by making pressure on important parts (dyspnea and interference with deglutition). The *diagnosis* is not difficult; it is based on their firm consistence and slow growth. The latter characteristic differentiates them from sarcoma. They should be *extirpated,* a measure which is difficult only when the tumor is large and its origin deeply located.

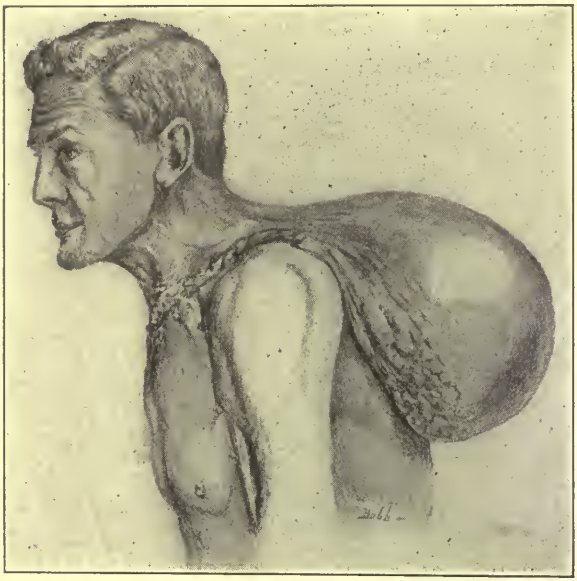

FIG. 821.— FIBROLIPOMA OF NECK.

Lipomata seem to show a predilection for the neck. The *circumscribed form* presents a characteristic sharp outline (see Part VI, chap. vii); however, occasionally a lipoma of this sort attains a very large size. *Subfascial lipomata* cause disturbances by pressure upon the trachea and esophagus. Those located in the supraclavicular region are likely to cause neuralgia (pressure on the brachial plexus) or obstruct the subclavian vein; the latter gives rise to edema of the arm. The *diffuse form* of lipoma (Madelung's[6] fat neck) is discussed in the general part of this book.

The *clinical picture* of lipoma is so characteristic that the *diag-*

nosis should be readily made. On the other hand, subfascial lipoma cannot be diagnosticated until it is exposed. Extirpation is not difficult, except in so-called fat neck, in which instance it is necessary to achieve the purpose in several sittings (Debrez[7]).

Neuromata of the superficial tissues of the neck do not present any regional peculiarities. Of the *deeply located neuromata*, those originating from the *vagus* are of importance. Jordan and Völcker[8] quote four cases collected by Semel and add one of their own seen in Lexer's clinic. Attention is called to the tumor by persistent hoarseness and the attendant cardiac disturbances. Removal of the tumor is followed by paralysis of the recurrent laryngeal nerve.

Enchondromata of the neck do not present regional peculiarities beyond the pressure symptoms.

Malignant Tumors of the Neck.— Malignant tumors of the lymph nodes of the neck include the general class of sarcomata. However, the process possesses so many histological differences that a *classification* in this connection is impossible. A rough clinical division into Hodgkin's disease, which is attended with enlargement of the lymph nodes in various portions of the body, and lymphosarcoma of a single node or cluster of nodes is as far as one is justified in going at this time. Both of these processes are taken up in Part VI, chap. xi.

Lymphosarcoma in the neck is not common. It appears in the form of a smooth, soft, movable, painless tumor covered with normal skin. As the growth enlarges, smaller nodules appear at its periphery, which soon merge with the primary tumor, until a large nodular mass is created. After a time, the growth becomes adherent to the skin, the muscles, and the deep vessels and nerves, and causes pressure upon the trachea and esophagus. As the process progresses, the skin breaks down and the protruding tumor undergoes disintegration. The entire course of the disease rarely extends over a year and a half. Strange to say, lymphosarcomata of the neck do not frequently form metastases.

The *treatment* is very unsatisfactory. Early extirpation of the growth holds out the only hope of relief. The use of the X-ray should not be withheld, although its therapeutic value is, to say the least, doubtful. The employment of serum is taken up in Part VI, chap. xi.

Primary carcinoma of the neck, involving the skin, is uncommon; it does not possess any regional peculiarities. *Branchiogenic carcinomata* begin in the deeper structures of the neck and are believed to

originate from residual epithelial cells displaced during embryonic development.

An exact *diagnosis* is impossible. On the other hand, the malignant character of the process should not escape recognition. It is important to differentiate it from growths that are secondary to hidden carcinoma

FIG. 822.— LYMPHOSARCOMA OF THE NECK.

of the nose, larynx, pharynx, etc. Differentiation from carcinoma of aberrant thyroid glands is possible only with the aid of the microscope. As in all malignant growths, sarcoma, tuberculosis, actinomycosis and lues must be taken into account (Fredet, Labay, Chevassu,[9] Forque et Massabuau,[10] Duthre,[11] Duret[12]).

Teratomata are usually situated in the side and anterior portions of

the neck. The diagnosis is usually made after *extirpation* (Niosi,[13] Hardouin,[14] and also Part VI, chap. xxii, of this book).

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CHAPTER V

SURGERY OF THE LARYNX AND TRACHEA

Examination of the air passages entered a new era when the now so familiar laryngeal mirror was introduced (1858) and the usefulness of the method was enormously enhanced by the introduction of cocain anesthesia (1884). While laryngology is largely in the hands of the specialist, the problems met with in this connection are often of a nature requiring the coöperation of the surgeon; a desideratum perhaps not as fully realized as it should be. However, the picture presented to the examining eye by the mirror is indicative of the condition of only a narrow area of the larynx, and is of course much distorted. This has led to the development of *direct laryngoscopy* which has made great strides since Kirstein[1] first published his method in 1894. The next step in this connection was presented by Killian,[2] who devised *upper bronchoscopy,* and later developed *direct bronchoscopy.*

Direct inspection of the air passages is based on the introduction of a straight metal tube, *per vias naturales,* into the bronchi. The technic of the procedure has been developed by Brünings,[3] who devised the *instrument* shown in Figure 823. It consists of two telescoping tubes. The outer terminates in a taper, which facilitates its introduction into the larynx; the inner is introduced through the outer and is held in place by a spring which prevents its expulsion. *Light* is furnished by an electrical bulb and condenser which are attached to the proximal end of the apparatus. During the examination the patient may be postured in the sitting (Fig. 823) or the supine position; the latter is preferable. The mucosa is cocainized. Albrecht[4] has devised a "swivel spatula" which makes additional exposure of the air passages possible. With this instrument it is feasible directly to visualize the trachea and both bronchi (*tracheography and bronchometry*). Low bronchoscopy through a tracheotomy wound, except perhaps for removal of a foreign body, is no longer necessary.

Malformations of the Larynx.— Malformations of the larynx are

practically limited to adherence of the *epiglottis* to the base of the tongue, and to *congenital diaphragm of the larynx.*

Congenital diaphragm of the larynx is of surgical importance. The glottis is more or less obstructed by a membrane located between the vocal cords. The condition is ascribed to incomplete patency of the rudimentary trachea. The *treatment* consists in excision of the adventitious membrane. In most instances this may be accomplished by the

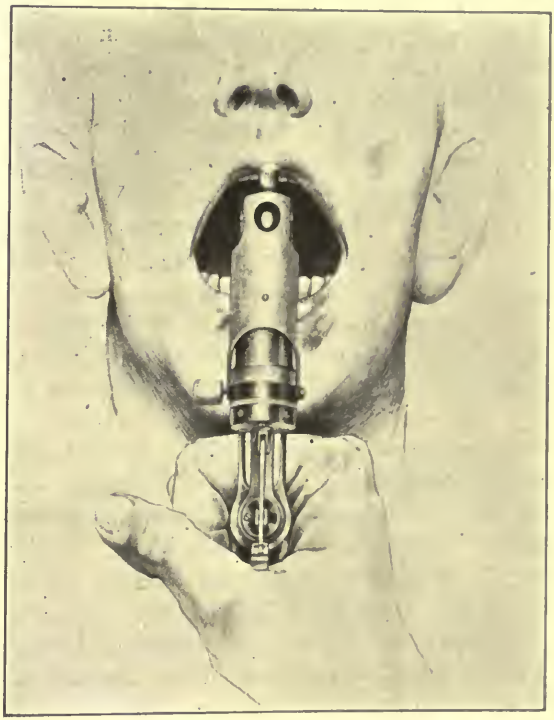

FIG. 823.— POSITION OF THE UNIVERSAL ELECTROSCOPE (BRÜNING'S) WITH THE PATIENT SEATED.

endolaryngeal route; however, in a certain number of cases, preliminary *laryngotomy* is necessary (Hansberg[5]).

LARYNGOCELES AND TRACHEOCELES (Arcocles).— Although generally ascribed to traumatic causes, there is no doubt that areoceles are due to at least a predisposing (congenital) malformation favoring their development.

The *congenital form* consists of a hernial protrusion of the mucosa. *Laryngocele* (single or double) is situated at the side of the thyroid

cartilage. The *diagnosis* is based on the fact that the sac is distended during expiratory efforts and may be emptied by pressure. *Tracheal protrusions* may be mistaken for cysts of the thyroid gland. In some instances the exact condition is not recognized until the protrusion is exposed (Petit[6]).

Concussion of the Larynx (Commotio laryngis).— Severe dyspnea, syncope and death have occurred in connection with trauma applied to the larynx without the presence of demonstrable solution of continuity. Theoretically, the employment of artificial respiration and, possibly, tracheotomy are indicated (Lubet-Barbon[7]).

Fracture of the Larynx and Trachea.— Fracture of the larynx is produced by *lateral compression* or by *flattening of its sagittal diameter* by pressure against the vertebral column. Of the accidental causes, a fall, a blow, or strangulation are the most frequent (see Hopmann's[8] collection of 145 cases). Most of the fractures are transverse; however, the solution of continuity of the cartilage may be in any direction and is not infrequently comminuted and even compound. The associated injury of the soft parts is likely to be extensive. Effusion of blood occurs within the larynx and also infiltrates the surrounding tissues. Edema may obstruct the lumen of the larynx to a dangerous extent.

The *symptoms* are those of a severe injury. In almost all instances, consciousness is temporarily lost (commotio laryngis). *Cough,* usually attended with bloody sputum, is a very frequent occurrence; *pain* is severe, especially when coughing or swallowing; the *voice* is rough, hoarse or absent. Primarily, respiration is much disturbed; then is less so; later is still more embarrassed; and soon develops stridor. The patient becomes cyanotic and is apprehensive of choking to death; the pulse is small, the skin pale and moist. As the edematous and bloody infiltrate of the mucosa increases, the respiratory difficulties are amplified. When the mucous membrane is ruptured, the surrounding cellular tissues, and later those of nearly or quite the entire body, are invaded by emphysema (Part III, chap. i). To this must be added the classic symptoms of fracture. Despite the menacing picture, the prognosis is not very unfavorable. A *fatal outcome* is caused by suffocation due to edema, accumulation of blood in the lungs, mediastinal emphysema, and aspiration pneumonitis. *Repair* takes place by callus formation. Late disturbances are ascribable to *reparative narrowing* of the lumen of the larynx.

The *treatment* aims at maintaining the free passage of air into the

lungs. As soon as dyspnea occurs, *tracheotomy* must be performed. However, the dyspnea may be caused by bleeding in the interior of the larynx, in which event tracheotomy is of no avail. In these cases, preliminary tracheotomy, median laryngofissure and tamponade of the larynx and repair of the structures in layers is indicated (Wagner[9]).

Fracture of the trachea is caused by the same influences discussed in connection with the larynx. The solution of continuity may be *complete* or *incomplete;* usually it is located just beneath the larynx or at the tracheal bifurcation. The *clinical picture* is very similar to that presented in connection with fracture of the larynx. The *diagnosis* is based on the fact that this is presented while the larynx is intact. The *prognosis* is unfavorable. The *treatment* follows the same principles applicable to the larynx. To this may be added, that dyspnea should not be awaited before operative efforts at repair of the lesion are made (Hansberg[5]).

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CHAPTER VI

WOUNDS OF THE LARYNX AND TRACHEA

Gunshot Wounds.— Gunshot wounds of the larynx are really complicated fractures of this organ, which is usually splintered, and are attended with more or less trauma to the vocal cords. The *clinical picture* and the *treatment* of the injury are similar to those discussed in connection with fracture.

Incised and Stab Wounds.— Incised and stab wounds of the larynx are most often *self inflicted* or are the result of attempted murder. The former are usually transverse and may involve the hyothyroid membrane, the thyroid cartilage, or the cricothyroid space. Wounds of this sort gape widely; occasionally inspiration draws the lower flap into the larynx and obstructs respiration. Dyspnea may also be caused by the flow of blood into the trachea, submucous hematoma, edema and abscess formation.

The *diagnosis* is difficult only in cases of stab wounds of the trachea, in which event the presence of bloody expectoration and the occurrence of emphysema decide the question. *Healing* is slow, even after repair by suture (20 to 30 days).

In the *treatment of wounds of the larynx and trachea* the chief factors are to maintain the *free passage of air* to the lungs and to arrest *bleeding*. Blood that has flowed into the bronchi may be removed through a sterile catheter by suction. In all cases, *prophylactic tracheotomy* is imperative. If the larynx is splintered and the soft parts severely traumatized, the wound should be cleansed and treated by the open method. On the other hand, in cases presenting less severe injury, an effort should be made to obtain union by primary intention. However, as this is not frequently crowned with success, ample provision for drainage must be made (Platt[1]). While circular repair by suture of the completely sectioned trachea has been successfully carried out on several occasions, the external wound must be drained and a simultaneous tracheotomy performed. As a rule, gunshot wounds are best treated by the open method (Boljarski[2]). The sequential cicatricial stenoses are taken up later (p. 1737).

Burns.— Burns and chemical cauterization of the larynx and trachea are the result of aspiration of heated air (or the flame) or of the flow of heated or caustic (acid or alkaline) fluids into the air passages.

The *clinical picture* is that of edematous swelling of the tissues, especially in the zone of the epiglottis and the aryepiglottidean folds. However, this may extend into the larynx and trachea, particularly when the condition is due to a caustic fluid. The chief *symptoms* are dyspnea, painful deglutition and coughing; the last is especially distressing. The *treatment* consists in tracheotomy and the modern methods of local anesthesia. Impregnation of the inspired air with moisture contributes very much to the patient's comfort (Pitts,[3] Hopmann[4]).

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CHAPTER VII

FOREIGN BODIES IN THE AIR PASSAGES

Most foreign bodies gain access to the air passages *per vias naturales,* although occasionally one enters through their walls (projectile, needle, tracheotomy tube, sequestrum from one of the vertebrae). As a rule, the entrance is precipitated by a *sudden forced inspiration* caused by fright, laughter or a fall. The presence of *disturbances of motility* of the larynx, the sequelae of a pathological condition (diphtheritic or bulbar paralysis, chronic inflammatory processes, etc.) favor the entrance of articles of diet and fluid into the air passages. The aspiration of foreign bodies is favored by *abolition of the reflex excitability of the larynx* (sleep or narcosis). The character of the foreign body varies greatly; the most common are false teeth, tobacco cuds, bonbons, and coins. V. Bruns and v. Hofmeister[1] present an interesting review of the literature in this connection.

The *localization* of the foreign body also varies, and in a measure is decided by its size. Of 646 cases collected by Heller,[2] 132 were in the larynx, 113 in the trachea, 161 in a bronchus, and in 240 the exact location is not stated.

A practicable division may be made into *movable* (fluttering) and *immovable* bodies (impacted). The former are found only in the trachea and bronchi; those located in the larynx are always immovably fixed in the sinus of Morgagni or in the glottis. Many bodies are arrested at the bifurcation of the trachea. As no air gains access to the portion of the lung obstructed by the invader, there is no expiratory *vis a tergo,* consequently, inspiration only serves to more tightly impact it (Gottstein[3]).

The changes in the tissues produced by the presence of the foreign body vary with its location and character and with the length of time it remains *in situ.* In the larynx, the protracted presence of the offending agent is followed by *strong reactive inflammation,* or pressure necrosis develops at once. These processes are both attended with edema of the surrounding tissues (especially the glottis). Necrotic

destruction of tissue often extends into the deep tissues, perforates the larynx, and invades the deeper structures of the neck. In some instances, the perforation extends into the esophagus; in others (very few), a perilaryngeal abscess is formed, which ruptures and the foreign body is discharged. Erosion into the large blood vessels and fatal bleeding has occurred. In other cases the reaction is limited to proliferation of granulation tissue. A small body, such as a needle, may become encapsulated.

A most severe reaction is developed in connection with the *entrance of a foreign body into the deeper air passages.* This is followed by trachitis and bronchitis with purulent secretion, inflammatory and cicatricial stenoses, recurring bronchopneumonitis, and abscess and gangrene of the lung. Occasionally, a foreign body perforates into the pleural sac and empyema develops. If a bronchial lumen is entirely occluded, the distal portion of the lung undergoes so-called obstruction atelectasis, and if pneumonitis is not developed, the occluded sector undergoes "collapse induration," while the surrounding lung tissue becomes emphysematous.

The *clinical picture* varies with the seat of the foreign body.

In the larynx, the invader provokes a stormy attack of coughing attended with a feeling of strangulation, cyanosis of the face, anxious expression of countenance and aphonia. After a time, this occurs in paroxyms with intervals of comparative quiescence. During the quiescent period, respiration is more or less embarrassed. The expectoration is at first bloody, and later purulent in character.

When a foreign body gains access to the *trachea and bronchi,* the picture depends upon whether the invader is *movable or immovable.* Of the subjective symptoms, dyspnea is the most marked. *Physical signs* are of the greatest value. At times the palpating finger discovers the foreign body in the trachea or the stethoscope reveals a fluttering sound (*grelottement* of Dupuytren) in this situation. If the greater part of a bronchus is occluded, the respiratory murmur of the lung supplied by it is diminished or absent. The respiratory excursions of the affected side of the thorax are diminished; vocal fremitus is lessened (Weist[4]).

The *diagnosis* is based on the history of the case, the physical signs, and the findings of the *laryngoscopic mirror and direct laryngoscopy.* Of these, the latter, under chloroform narcosis, is the most reliable. In addition to this, the *Röntgenographic* negative is an exceedingly valuable aid (Fig. 824). Its value, of course, is dependent upon the

character of the foreign body; however, even though the object is not reproduced, the changes in the outline of the lungs, as seen on the negative, are of diagnostic value.

The *prognosis* may be said to be very unfavorable, unless the foreign body is removed, and is worse the longer this is postponed.

In the *treatment*, irrespective of the views of the individual sur-

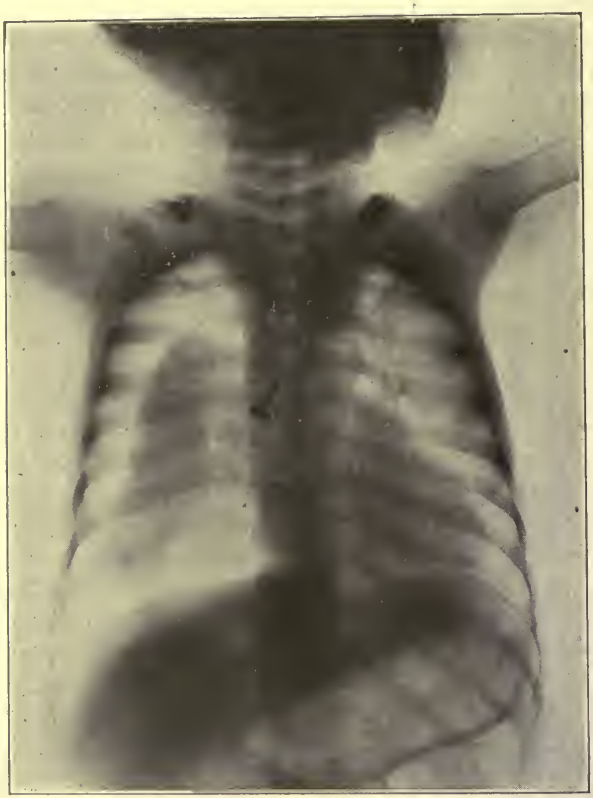

FIG. 824.— RÖNTGENOGRAM OF A FOREIGN BODY (TACK) IN THE RIGHT BRONCHUS (Harlem Hospital case).

geon, none will deny the imperative necessity for tracheotomy when death from strangulation is threatened. The exception to this is when a foreign body is located at the entrance to the larynx and can be reached with the finger. The expectant plan of treatment is to be condemned; the use of emetics is of doubtful value. If the condition of the patient permits, an effort may be made to locate the offending agent with the mirror and to extract it with suitably fashioned forceps;

failing in this *direct laryngoscopy, tracheotomy and bronchoscopy* are taken into consideration.

The indications for tracheotomy in connection with deeply located foreign bodies have been somewhat restricted by the advent of the bronchoscope. Killian[5] regards the indications for tracheotomy as being when great dyspnea exists, when lung complications have developed, when the foreign body is large and so fashioned that its removal through the mouth would be likely to do damage, and when the invader consists of a substance likely to swell and become impacted (bean). The presence in the trachea of a fluttering object is an indication for immediate tracheotomy. On the other hand, the technic of bronchoscopy is by no means easy; the "opportunity bronchoscopist" is a dangerous person. Foreign bodies located in the *larynx* should be removed by *tracheotomy;* if the body is impacted, *laryngotomy* should be practiced. *Subhyoid pharyngotomy* possesses no advantage over the latter. Tracheotomy should also be employed for the removal of foreign bodies located in the trachea, unless the conditions are particularly favorable to rapid and skillful employment of the bronchoscope.

Bronchoscopy finds its greatest field of usefulness when the foreign body is located at or below the bifurcation of the trachea. For the purpose, the universal electroscope of Brünings (Fig. 823) is largely used to locate the invader and a number of ingenious forceps and snares are employed to effect the extraction. These are too numerous to describe here.

In 1913 Mann[6] of Dresden said "The first book regarding tracheobronchoscopy, in the English language, originated with Chevalier Jackson[7] of Philadelphia. In this the specialty received the impress of the individual. The inventive spirit of the American and his great personal independence, which knows not *iurare in verba magistri,* may be the reason for the improvements in technic which emanate from his country." Almost a prophecy.

The complicated transmediastinal bronchotomies performed so far have not given encouraging results.

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CHAPTER VIII

INFLAMMATORY DISEASES, STENOSES AND NEUROSES OF THE LARYNX AND TRACHEA

In inflammatory diseases of the air passages the function of the surgeon consists in the maintenance of the passage of air to the lungs rather than the treatment of the disease itself; the latter belongs to the internist and the laryngologist. Of the diseases belonging to this class, *diphtheria* is the most important; although this has already been extensively discussed (Part II, chap. xvi), the purely surgical aspects of the problem are still to be taken up.

While *stenosis of the larynx* is the chief symptom, in itself pleading, piteously enough, for *tracheotomy,* there is also no doubt that the incision has a favorable influence upon the circulatory disturbances coincident to the disease and that it is a VALUABLE PROPHYLACTIC MEASURE AGAINST BRONCHOPNEUMONITIS (Rauchfuss[1]). If tracheotomy is postponed until respiratory embarrassment is menacing, the prognosis is much more unfavorable (Krönlein[2]).

A discussion of the relative merits of tracheotomy and intubation does not belong here. The conclusions in this connection seem to be that the latter method has certain serious objections in diphtheria, the chief one being that an already infected mucosa is additionally damaged. In the hands of the trained operator the technic of intubation is not difficult; however, extraction of the tube is not so readily accomplished. The instrumentarium is depicted in figure 825.

Intubation of the larynx by the O'Dwyer method may be described as follows: The child is seated on the lap of the nurse; an assistant steadies the head and slightly extends it; the mouth is held open by the gag; the left index finger pulls the epiglottis and the base of the tongue forward; the cannula is passed along the posterior surface of the epiglottis; the introducer is withdrawn while the finger prevents withdrawal of the cannula. The safety string is either removed or fastened to the patient's ear. The introduction of the *extubator* is carried out in the same way. In infants the tube may often be expressed by making upward pressure on the external surface of the larynx.

Edema Laryngis; Laryngitis Submucosa.— Edema of the larynx is most often the outcome of an inflammatory process. Differentiation between an edema appearing as a part of general disturbances, such as anasarca or the result of venous stasis (tumors of the neck and mediastinum), and that which is the result of a genuine inflammatory process is not always possible from a clinical standpoint. However, the latter form of the affection is more properly designated as *laryngitis submucosa* (Küttner[3]). While several more or less complicated classi-

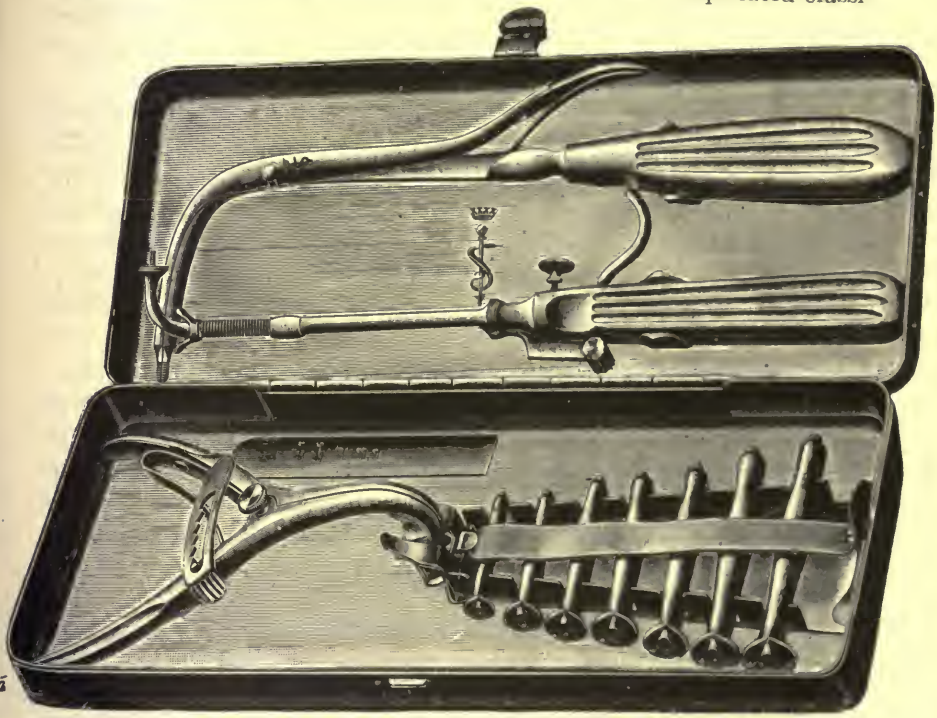

FIG. 825.— O'DWYER'S INTUBATION SET.

fications of the processes have been submitted, that of Hajek[4] is the most simple and is based on its etiology as follows: (1) Those occurring in connection with local affections of the larynx and its vicinity, i. e., *symptomatic* and *extension edema;* and (2) *infectious edema* which develops primarily in the submucosa of the larynx or occurs in the course of an acute infectious disease.

The *first group* includes submucous inflammations, already taken up in connection with injuries, foreign bodies, chemical and thermal influ-

ences, diphtheria, and the various infections of the pharynx, tonsils, palate, etc.

The *second group* encompasses septicopyemic general infections, such as ulcerative endocarditis, erysipelas, typhus, variola, scarlatina, etc. It is probable that submucous laryngitis may be due to extension of an erysipelatous process from the face.

The submucous swelling may involve the entire larynx or may be limited to certain sectors. It does not present any regional peculiarities and is of special importance only because it obstructs breathing. Abscess formation involves the danger of sudden death by strangulation.

Of the *symptoms* of edema of the glottis, interference with the passage of air to the lungs is the most important, especially since this is likely to develop with great rapidity. The greatest difficulty occurs during inspiration, the result of the valvular action of the swollen arytenoid folds. The interference with vocalization is not characteristic. The most rapidly developing form of the affliction occurs in connection with erysipelatous and acute infectious phlegmon (Gerber[5]).

The *diagnosis* of edema of the larynx is easily made with the mirror; however, extensive swelling of the aryepiglottic folds often obscures the view into the larynx itself. Under no circumstances must efforts to visualize intralaryngeal swelling be attended with ungentle manipulations. As a rule, the diagnosis may be based on the history of the case and, if this is not obtainable, an exact conclusion in this connection may very properly be postponed until the dyspnea is relieved.

The *treatment* is primarily directed toward preventing the edema in cases in which this complication is likely to occur. Of these, the swallowing of particles of ice is of great benefit. Astringents should be avoided. The dominant indication in the problem is the maintenance of the free passage of air into the lungs. This is of course best established by prompt *tracheotomy* which, after all, is the only measure capable of defeating what v. Ziemssen,[6] back in 1876, called "this perfidious affliction."

In cases of stenosing laryngeal catarrh in children (pseudo croup), the use of O'Dwyer's intubation method may be regarded as distinctly indicated.

Laryngeal Perichondritis.— Inflammation of the laryngeal perichondrium is usually *secondary* and occurs in connection with ulcerative processes of the mucosa (tuberculosis, lues, typhoid, variola, carci-

noma, etc.) which allow of the passage of pyogenic bacteria, or the process is metastatic in the course of general infections (scarlatina, typhoid fever, etc.). Perichondritis also develops after trauma to the interior of the larynx (foreign bodies, chemical burns, etc.). *Primary perichondritis* is a term applied to those cases in which the port of entrance of the provocative influence is not discoverable. The *anatomical changes* are similar to those taken up in connection with perichondritis and periostitis (Part II, chap. x). Of the various parts of the larynx, the arytenoid cartilages are most often affected. Similar processes occur in the *trachea,* but they are rare.

The *dangers* of perichondritis of the larynx consist in mechanical obstruction to respiration, either from the inflammation or from collateral edema. Asphyxiation from the incarceration of a loosened sequestrum has occurred. *Secondary stenosis* and permanent fixation of the vocal cords, the result of sequential cicatrical contracture, is not uncommon.

Painful deglutition, hoarseness and dyspnea are the chief *symptoms.* However, the *diagnosis* must be established by laryngological examination and the symptoms of inflammation.

The *treatment* is similar to that used in connection with inflammations in general. The indication for tracheotomy rests on the degree of dyspnea; it should not be delayed. Incision of perichondritis externa is of course accomplished from without. *Endolaryngeal incision* of processes bulging into the larynx is a difficult maneuver unless the abscess is located very high up. On general principles, these should also be opened from the neck. As in all affections of this nature, a policy of timidity is to be deprecated. Laryngotomy and the establishment of drainage, with the bronchi protected by a tracheotomy tube, gives the best results.

Specific Inflammations of the Larynx and Trachea.— The primary and secondary chronic inflammatory lesions of the larynx and trachea usually are either tuberculous or luetic in nature. A few of the cases are due to the persistence of a so-called catarrhal inflammation, which no doubt is also infectious in character; the precise nature of this infection is not known. These have been burdened with a number of names, such as *chorditis vocalis inferior hypertrophica, blennorrhea, laryngoscleroma,* etc.

Tuberculosis.— *Tuberculosis of the larynx* alone is very rare; in the vast majority of cases it is *secondary* to a similar lesion situated elsewhere in the body, usually the lung.

The character of the lesion does not differ from those located elsewhere in the body. The vocal cords and the posterior wall of the larynx are at first involved. The fact that the cord first affected corresponds to the invaded lung is worthy of note. The process becomes of surgical importance when it is attended with *deep ulceration*, invasion of the perichondium and edema.

Tuberculosis of the trachea occurs in one half of the autopsied cases of laryngeal tuberculosis.

The *diagnosis* is based on dysphagia, aphonia, cough and pain (the last is likely to be severe), together with the laryngological findings; however, it is only certain when the bacilli are found in a smear taken from the lesion. The coexistence of tuberculosis and syphilis in this situation is not infrequent. The *prognosis* is unfavorable, the fatal outcome being ascribable to the underlying condition (lung).

The *surgical treatment* consists in the local application of anodynes (anesthesin, orthoform, alypin). The injection into the superior laryngeal nerve of 85 per cent alcohol produces anesthesia of the larynx for from six to forty days and is a justifiable measure of relief. Severe dysphagia justifies *gastrostomy*.

Direct attack by the external route brings the affliction into the domain of the general surgeon. *Endolaryngeal operative measures* are not often productive of satisfactory results. *Tracheotomy* is indicated when edema causes menacing obstruction. *Laryngectomy* (complete or incomplete) is a bold undertaking; however, complete recovery followed in four of twenty cases operated upon by Gluck[7]. *Laryngofissure* and excision of the lesion has been practiced more than one hundred times. The local recovery has been much shadowed by the ultimate domination of the lung condition. The cases in which the procedure is permissable must be carefully selected. Exacerbation of the process in the lung has been frequently observed.

Lupus of the larynx occurs in the form of *nodular* and *papillomatous excrescences*. It is likely to simulate lues and lepra in this situation (Mayer[8]).

Syphilis.— *Lues of the larynx* appears at all periods of the general disease and occurs in 10 per cent of the cases (Gerhardt[9]). The *ulcerative form* is ascribed to disintegrated gummatous infiltration. This usually denudes the perichondrium and at times destroys the cartilage. Healing is attended with cicatricial stenoses.

In the *diagnosis*, tuberculosis and carcinoma must be considered. One must bear in mind that two of these may be present in the same

individual. The therapeutic test is of some value in cases of this sort. In extensive lesions ("florid lues") emergency tracheotomy may become necessary. During repair the careful use of the bougie may obviate laryngeal stenosis. For the general treatment see Part II, chap. xxv.

Actinomycosis of the larynx has been found to be more frequent since the advent of the more general employment of the microscope in arriving at a conclusion in regard to the exact nature of chronic inflammatory conditions in the upper air passages. As a rule, the invasion of the larynx by the process is *secondary* to a lesion of the neighboring parts. *Primary* actinomycosis of the larynx does not seem to have been reported as yet (Henrici[10]). The *treatment* is similar to that employed in actinomycosis located elsewhere in the body. The local injection of bichlorid of mercury solution seems to be of benefit.

Scleroma of the larynx is rarely primary (see Part II, chap. xxvi; also Pieniazek,[11] and v. Rüdiger-Rydygier[12]).

Stenoses of the Larynx and Trachea.— *Stenoses of the larynx* occur in connection with a number of pathological processes in this situation; they may be divided into *temporary and permanent*. The former have already been considered.

The *etiology of permanent stenosis of the larynx* has been discussed in connection with the pathological processes occurring in this situation. Almost all of these may be responsible for narrowing of the lumen of the larynx, while a few are always followed by it. Of the causative lesions, those occurring in connection with *injuries, tertiary lues, and perichondritis* are the most frequent offenders. Perichondritis is most likely to be followed by stenosis when the former develops in the course of acute infectious diseases, such as typhoid, scarlatina, and variola, in the order mentioned. Late stenoses, following diphtheria, are due to trauma caused by intubation and tracheotomy, although the obstruction following the latter is located in the trachea. Narrowing of the lumen of the larynx, sequential to intubation, is especially obstinate in character.

The *form of the stenosis* varies with its cause, the *diaphragmatic and funnel shaped* strictures being the most frequent, although the *cylindrical* form is not by any means rare.

Dyspnea and stridor are the *cardinal symptoms* of laryngeal stenosis and are manifested in proportion to the degree of the obstruction. It is astonishing how narrow an orifice will permit of the passage of suffi-

cient air to maintain life, especially if the obstruction develops gradually.

The immediate indication in the *treatment* calls for *tracheotomy;* this must not be postponed until the danger of strangulation becomes imminent.

The measures employed for the purpose of overcoming the stricture may be stated as follows:

1. *Intralaryngeal dilation without tracheotomy.*
2. *Dilation after tracheotomy,* using the wound as a means of approaching the stricture.
3. *Laryngotomy.*
4. *Resection of the larynx.*

Fig. 826.— Cicatricial Stenosis of the Larynx Following Suicide Wound (viewed from behind).

Dilation may be assisted by endolaryngeal discission of the stricture; this is especially applicable when the latter consists of a thin membrane. The extralaryngeal methods must always be followed by a more or less prolonged use of dilators (Rosenberg[13]). *When a tracheotomy wound is present* the dilators may be passed from below and withdrawn from the mouth. In these cases hollow dilators may be left *in situ* for varying periods of time.

Laryngofissure is employed for the purpose of introducing dilating apparatus (T-cannula, chimney cannula, etc.)., or to permit of the *excision of cicatrical tissue.* By this method of approach *subperichondral resection* of the cartilage of the larynx may be performed. In recent years a *laryngotomy* is first performed and the fistula is used for subsequent dilation of the stricture. In this method the opened larynx is fastened to the skin, which permits of the use of variously shaped dilators; these may be left *in situ* for days at a time (Sargnon et Barlatier[14]). The fistula is ultimately closed by means of a flap.

Gluck[15] excises the stricture and turns two skin flaps into the larynx. The dilatation method must be persisted in for months.

Dilatation in connection with tracheotomy permits of retention of the dilator without danger of suffocation, and in most cases will be found a satisfactory method. In the hands of the trained technician *laryngofissure* and excision of the stricture and plastic repair give the most brilliant results.

Stenoses of the trachea are often caused by extramural pressure, of which enlargement of the thyroid gland is not the least important. The *intratracheal causes of stenosis* (exclusive of those provoked by a tracheotomy tube) are similar to those operative within the larynx, and, of these, lues again plays the leading rôle.

The *general symptoms* of tracheal stenosis do not differ from those of laryngeal obstruction. The *diagnosis* will have to rest very largely upon the history of the case, and of course direct *bronchoscopy* will make it certain. The Röntgenogram is of great assistance. Rethi[16] introduces a photographic film into the esophagus and obtains remarkably clear shadows of the trachea.

The *treatment* in many respects is similar to that discussed in connection with the larynx. Low obstructions are reached with specially constructed instruments introduced through a tracheotomy wound. Sectional resection of the stenosed area and end to end suture is permissible in selected cases (v. Eiselsberg[17]).

Fistulae of the Larynx and Trachea.— Fistulae of the larynx and trachea are the result of injury and of necrosis. Those of the latter class are subdivided into *ulcerative* and *cicatricial.*

Ulcerative fistulae follow suppurative perichondritis, diphtheria, carcinoma, etc. *Cicatricial fistulae* appear in connection with ulcerative processes (other than carcinoma) which do not entirely heal, and when the mucosa becomes adherent to the skin. Either form of fistula may be located in any of the areas of the larynx or trachea. In the cicatricial form the fistula usually corresponds to the point of greatest stenosis.

The external skin is usually drawn inward in the form of a funnel, at the apex of which the opening in the trachea is visible. If stenosis is located above the fistula, closure of the latter must not be attempted.

The significance of an air fistula depends upon its size. The smaller ones are simply an annoyance, while the larger, if associated with stenosis, condemn the patient to the perpetual use of a cannula.

An air fistula is often associated with an esophageal opening in cases

of severe injury in this location, especially after transverse section of the upper trachea and esophagus. However, persistence of the esophageal opening is rare.

The *disturbances* caused by a communication between the trachea and osephagus relate especially to the entrance of food into the former. The *diagnosis* is easily verified with the esophagobronchoscope.

Closure of an air fistula is permissible only in the *absence of stenosis of the air passage.* The operative methods of relief are grouped under the name of *bronchoplasty.* Small fistulae may be closed by the use of the cautery and a purse string suture, or complete excision and repair by suture may be practiced. The larger ones must be closed by plastic methods. A free skin periosteal flap, taken from the sternum, was successfully implanted by Schimmelbusch.[18] Photiades and Lardy[19] used a pedunculated flap from the clavicle. A tracheal fistula may be closed by a cartilage flap turned down from the larynx (Koenig[20]).

The *neuroses of the larynx* possess surgical importance only when *bilateral paralysis of the vocal cords* is attended with obstruction to breathing. In these instances *tracheotomy* is imperative.

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CHAPTER IX

TUMORS OF THE LARYNX, TRACHEA, AND PRIMARY BRONCHI

Most of the tumors of the interior of the larynx and trachea belong to the domain of laryngoscopic surgery, which has accomplished much in this connection. While this is true of the majority of *benign tumors,* it is also true that the problems connected with *malignant tumors* belong to the field of general surgery. The subject will therefore be taken up from this standpoint.

Benign Tumors of the Larynx.— Fibromata and papillomata are the tumors most frequently found in the larynx. In 370 cases, 55 per cent were fibromata and 35 per cent papillomata (v. Bruns[1]).

Fibromata develop in the submucosa and vary in size from a pea to a cherry. They are smooth (occasionally nodular), have a wide base, are dark red in color, and are usually located at the anterior edge of the vocal cords. They develop late in life.

Papillomata appear in the form of warts or berries, and at times resemble the slender condylomata of the skin. Collections of a number of these growths take on peculiar forms. They are red in color and, when covered with horny epithelium, may be gray.

The importance of papillomata lies in the fact that they appear early in life and are likely to undergo malignant changes (carcinoma).

Cystic tumors of the larynx are very rare (4 per cent). *Myxomata, angiomata, and lymphangiomata* are also uncommon (Chiari[2]). *Lipomata* may reach a large size. As is to be expected, *chondromata* are not so rare; however, they are by no means common.

The *symptoms* of benign tumors of the larynx depend upon their size and location. Most of them are situated upon the vocal cords, including the anterior commissure. Of 1,000 cases seen by v. Bruns,[1] 72 per cent showed *modification of the voice.* Aside from this, a number of small tumors of the vocal cords simply cause modification of the singing voice (*"singer's nodules"*). The degree of *interference with respiration* depends of course upon the volume of the growth;

it is not often great. *Pedunculated tumors* interfere with function intermittently.

The *diagnosis* can be made with certainty only by *laryngoscopic examination,* which determines the presence, extent, and often the character of the growth. However, the latter must be verified by means of the microscope.

Extirpation of benign tumors may often be accomplished by the *endolaryngeal route;* in a certain number of cases, *laryngofissure* is necessary. Execution of the former methods means special apparatus and considerable technical skill; it is quite restricted to the laryngologist. The employment of the direct method has made removal of heretofore inaccessible tumors possible and has the additional advantage of permitting general narcosis. Preliminary exposure by *laryngofissure* is indicated when the growth is attached by a broad base and is of large dimensions, or when a large number of papillomata are present. *Partial laryngotomy,* below the vocal cords, is desirable when the seat of the growth justifies its employment. *Subhyoid pharyngotomy* gives excellent access to the upper sectors of the larynx.

FIG. 827.— MULTIPLE PAPILLOMATA OF THE LARNYX.

Malignant Tumors of the Larynx.— Of the malignant tumors of the larynx, sarcoma, compared to carcinoma, occurs once in twelve cases.

Sarcoma of the larynx is essentially a disease of early life. In 1908 Hiltermann[3] presented a statistical study of 125 cases of primary and 17 cases of secondary sarcoma of the larynx. The tumor is usually situated at the vocal cords and shows little tendency to break down. It appears in the form of a sharply defined, hard, light red mass with a smooth surface, and usually is attached by a broad base.

It grows very rapidly, soon reaching the size of a walnut; however, its extension is limited to the soft parts, and erosion of the cartilage is rare. Lymphatic invasion occurs late and may be absent. Of the various forms of sarcoma, the spindle cell, which originates in submucosa, is the one most commonly found.

The *diagnosis* is not easy, especially as the growth at first bears considerable resemblance to a benign tumor. Microscopical examination of a section removed by the endolaryngeal method clears up the problem.

The *treatment* is limited to complete *laryngectomy.* Despite this radical measure, the process recurs in 80 per cent of the cases.

Carcinoma of the larynx occurs in the last half of life. Eighty-four per cent of the cases occur in persons more than forty years of age. It appears in all portions of the larynx; however, the rim of the thyroid cartilage (epiglottis) is most often affected. *Pavement cell* carcinoma is the most common form in which the process appears; adenocarcinoma is rare.

In the *early stages,* the disease appears in the form of a *circumscribed infiltrate* of the mucous membrane, which is flat or slightly elevated, or it presents the appearance of a warty excrescence. This condition of affairs may persist for several years.

In the *advanced stage* of the process the growth ulcerates and invades the contiguous tissues, losing its circumscribed character and being surrounded by an area of edematous swelling. The process now corresponds in every regard to that described in connection with carcinomata of mucous membranes (Part VI, chap. xvii). *Invasion of the neighboring lymph nodes* is not constant and appears late in the process, especially in the essentially endolaryngeal growths. *Metastases* are uncommon; however, the writer has seen vertebral foci in two cases in which laryngectomy has been performed.

Secondary carcinoma of the larynx is rare; on the other hand, it is often invaded by extension from neighboring parts.

The *clinical picture* of carcinoma of the larynx in its early stages varies with its location. When the region of the epiglottis is involved, *hoarseness* is a dominant symptom. v. Bruns[1] says "PERSISTENT HOARSENESS WITHOUT COUGH IN MIDDLE AGED MEN DEMANDS A LARYNGOSCOPIC EXAMINATION." The impairment of the voice is progressive and ultimately ends in aphonia. The latter is due to impairment of the vocal cords and to pressure on the motor nerves. So-called *extrinsic cancer* is heralded by *dysphagia,* which is progressive and may make

deglutition impossible. As the lesion progresses, dyspnea develops to a lesser or greater degree. *Pain* is always present. Later in the disease, bloody expectoration appears. Cachexia presents itself late.

The *diagnosis* is difficult in the early stages of the disease; it may then be established by the employment of all the accessory aids. These must assist in differentiating the lesion from lues, tuberculosis, actinomycosis, and benign tumors. One must remember that the last often become carcinomatous, especially papillomata. To the laryngologist, *rigidity of the affected vocal cord* is a suspicious sign. As to the extent of the lesion, the mirror is a deceitful helper, *direct laryngoscopy* is less so. Of course the *microscopical finding* should decide the question; however, Semon,[4] who warns us that this must be in accord with the clinical picture, states that of 246 cases observed in private practice, he failed to recognize carcinoma in fifteen. He furnishes an instructive résumé in this connection.

The *prognosis*, until thirty years ago, was very unfavorable and only comparatively better for the following twenty years. The improvement in the last ten years is the outcome of more exact diagnostic methods and the *technic of the radical operation*. The fact that endolaryngeal carcinoma is not attended with involvement of the lymph nodes early in the disease makes the prognosis less unfavorable in this class of cases.

The radical measures of relief may be stated as follows:

1. *Endolaryngeal extirpation.*
2. *Extirpation via thyroidotomy.*
3. *Extirpation via subhyoid pharyngotomy.*
4. *Partial extirpation of the larynx.*
5. *Total laryngectomy.*

1. Endolaryngeal total extirpation of carcinoma is possible, but may be said to be "taking an unwarranted risk," a view shared by most laryngologists of note.

2. *Thyroidotomy*, or laryngofissure, is the normal procedure in the early stages of endolaryngeal carcinoma. The mortality is 10 per cent (Bryson Delevan,[5] Santi,[6] Molinie[7]).

3. *Subhyoid pharyngotomy* is used only when the disease is located at the epiglottis and the aryepiglottic folds — so-called extralaryngeal carcinoma. The mortality is 40 per cent (Jurasz[8]).

4. *Partial laryngectomy* is indicated when the disease is limited to one side of the larynx (Gluck[9]). It has a restricted field of application, and a mortality of 26 per cent (v. Bruns[1]).

5. *Total laryngectomy* is indicated in cases in which partial extirpation is not. It is also performed as a part of the procedure when the disease has extended to the pharynx and esophagus. The operation achieves its most brilliant possibilities in cases of endolaryngeal carcinoma. It has a mortality of 22 per cent (v. Bruns[1]). The writer performs total laryngectomy as soon as the diagnosis is made.

Tumors of the Trachea.— Compared to tumors of the larynx, those of the trachea are rare; however, the advent of direct tracheoscopy has markedly lessened this difference.

Benign Tumors.— Of the benign tumors, fibromata and papillomata are the most common. Osteochondromata, as is to be expected, have also appeared in this situation (Thiesen[10]).

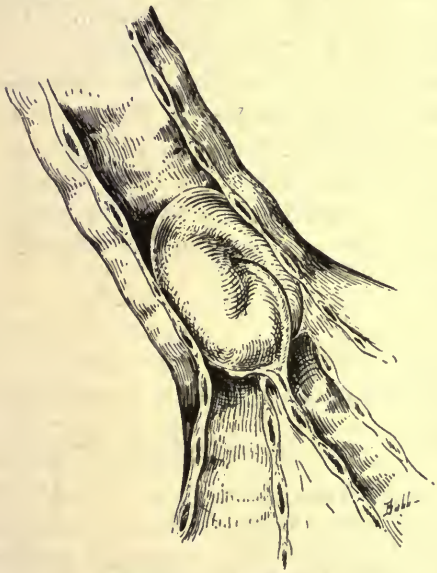

Fig. 828.— Lipoma of the Left Bronchus.

The *symptomatology* of benign tumors of the trachea may be encompassed within the clinical picture of obstruction to respiration, which is very gradual in its onset (Gerhardt[11]).

The *diagnosis* must exclude pressure on the trachea from without. In most instances the growth may be visualized by direct bronchoscopy.

The *operative treatment* has a number of brilliant results to its credit. The palliative treatment consists in providing a passage for air. This may be accomplished by low tracheotomy and the introduction of a long pliable tube.

Extirpation of the growth has frequently been accomplished through the bronchoscope, either directly or through a tracheotomy wound. The latter is the better method. V. Bruns[1] reports nine successful cases of this sort.

Malignant Tumors.— Malignant tumors of the trachea are not as rare as are the benign.

Primary sarcomata of the trachea are usually a part of a growth extending from the lower posterior portion of the larynx; most of them are spindle cell. Although a growth of this sort has occasionally been

removed through the larynx, approach by means of a tracheotomy wound best accomplishes the purpose.

Carcinoma of the trachea is more frequent than sarcoma, and usually involves the posterior, lower portion. The *diagnosis* is made by means of direct tracheoscopy. The outlook is unfavorable; however, resection of the affected portion of the trachea and end to end anastomosis has been successfully performed in several instances (v. Bruns[1]).

Tumors of the Primary Bronchi.— Exploration of the bronchi is so recent a measure that recognition of new growths in this situation is as yet restricted to a narrow field. V. Bruns[1] has collected seventy-five cases of tumors of the bronchi revealed by the bronchoscope. Of these, 36 were benign, 35 malignant, and 4 of unknown nature.

The *diagnosis* is of course possible only by means of the bronchoscope. This rather difficult examination is justified in the event of persistent cough and dyspnea, not explainable on the basis of the other numerous factors which are also responsible for these disturbances. There is no doubt that, were the possibility of a bronchial tumor more often taken into account, the number of cases would be considerably increased.

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CHAPTER X

OPERATIONS ON THE AIR PASSAGES

Tracheotomy.— In a general way tracheotomy is employed for two purposes — the ESTABLISHMENT OF A FREE PASSAGE OF AIR TO THE LUNGS and to PROVIDE ACCESS TO THE REGION BELOW THE GLOTTIS.

The special indications are as follows:

1. Injuries of the larynx and trachea.
2. Foreign bodies in the air passages.
3. Acute inflammatory processes in the larynx and trachea.
4. Chronic inflammatory processes.
5. Compression stenoses.
6. Neuroses.
7. Tumors of the larynx and trachea.
8. Preliminary tracheotomy in connection with operations on the upper air passages.

To these may be added the necessity for the measure in cases of *asphyxia* due to the inspiration of *gasses* and *chloroform.*

In the *technic of tracheotomy* it is necessary to take into account certain *anatomical considerations.* While the cricoid cartilage and the cricothyroid membrane do not actually belong to the trachea, they are included in the problem because of their frequent invasion when the trachea is opened. The trachea is superficially located at its upper aspect, but is gradually more deeply situated until at the episternal notch it is 3 cm. deeper than the skin. The cricoid ring is always palpable. The *isthmus of the thyroid gland* divides the tracheotomy field into two sectors: unfortunately the variations in its diameter do not permit of an exact localization in this connection. In addition to this the pyramidal lobe, which originates from the upper edge of the isthmus and extends upward, sometimes as high as the hyoid bone, must be taken into consideration. The pyramidal lobe is present in 74 per cent of the cases. The isthmus is held in contact with the trachea by a process of its own capsule (thyrolaryngeal ligament), which splits into two layers and is attached to the cartilaginous rings below. The

cricothyroid artery (a branch of the superior thyroid) crosses the membrane of the same name; below the isthmus a number of veins cross the trachea (plexus thyroideus impar); these communicate directly with the innominate vein. Occasionally the latter vessel and the artery obtrude into the episternal notch. *In children,* the space between the isthmus and the episternal notch is as long as in the adult, while that between the former and the cricoid cartilage is only one centimeter.

The space available for tracheotomy may be utilized as follows:

1. *Thyrocricotomy* — section of the conoid ligament..
2. *Cricotomy* — section of the cricoid cartilage, together with cricotracheotomy, in which one or more of the rings of the trachea are also divided.
3. *Superior tracheotomy* — opening of the trachea above the thyroid isthmus.
4. *Median tracheotomy* — section through the isthmus.
5. *Inferior tracheotomy* — section in the space between the innominate vein and the isthmus. From a practical standpoint, the operation is performed *either above or below the isthmus.*

Cricotomy presents the least technical difficulties.

The *posture of the patient* is important; the head is extended by placing a firm "roll" under the neck. Despite the prolonged discussion regarding the use of various anesthetics, this aspect of the problem may be answered by the unqualified statement that nothing takes the place of the *judicious administration of chloroform* when dyspnea is present. In the absence of the later, *local anesthesia* may be used *in the adult*

The *incision* is made in the median line and should be three or four centimeters in length; in any event, it should be ample rather than the reverse. The subcutaneous veins are divided between forcipressure, which act as retractors until the median borders of the sternohyoid muscles (linea alba colli) are identified. The intermuscular space is opened and the muscles drawn apart with retractors. In this way, the isthmus and tracheal rings are exposed and the point at which the trachea is to be opened is decided upon.

In *superior tracheotomy,* when the isthmus does not cover the upper end of the trachea, the deep fascia is opened in order to get the requisite room. If this is not the case (which is the rule), retrofascial separation of the isthmus is proceeded with; the thyrolaryngeal fascia is divided transversely at the lower edge of the cricoid cartilage and it, together with the isthmus, is lifted from the trachea and pulled down-

ward. If a pyramidal lobe is present, this is drawn down to either side or may be extirpated.

In *inferior tracheotomy* the deep fascia of the neck is divided below the isthmus. In manipulating in the pretracheal fat, the disturbing proximity of the innominate vein must be given consideration. The isthmus is pulled upward and the veins, already spoken of, are avoided. The transverse veins are clamped, divided and ligated.

Opening of the trachea must be *postponed until bleeding is definitely*

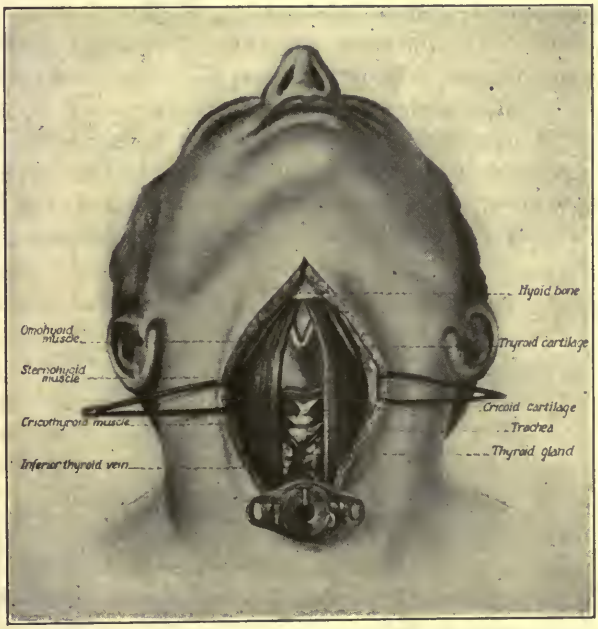

FIG. 829.— TRACHEOTOMY.

Showing spaces for various methods. Inferior tracheotomy completed.

arrested. The trachea is steadied with tenaculae introduced at either side of the media line, the ithmus is held out of the way, and the knife, with its cutting edge turned toward the chin, is plunged into the trachea and the incision made to conform to the size of the cannula to be used. As a rule, the sudden inrush of air is immediately followed by a *brief period of apnea,* the outcome of a sudden diminution of carbon dioxid in the medulla. The CANNULA IS NOT INTRODUCED UNTIL RESPIRATION IS TRANQUIL. Franck[1] advises transverse division of the

skin with the view of preventing scarring; this is its only advantage. Thost[2] advises excision of an elliptical sector of the trachea in order to obviate inversion of the tracheal rings. In *tracheotomie dans un seul temps* of St. Germain[3] the operator grasps the larynx with the left hand and plunges the knife into the cricothyroid space, dividing the cricoid cartilage and several rings of the trachea in one sweep. Its employment is justified only in cases of imminent strangulation.

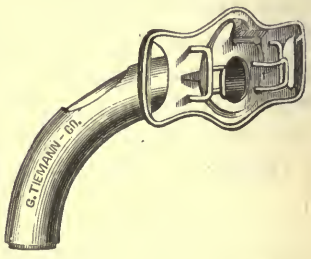

FIG. 830.—LUER'S DOUBLE CANNULA.

The *selection of an appropriate cannula* is of great importance. A description of the various cannulae exploited at different times would fill many pages. The writer still clings to the well known Luer double cannula fitted with a movable shield (Fig. 830); its curve corresponds to from one fifth to one fourth of a circle. The dimensions of a cannula suitable to the age of the patient are tabulated below:

Age	Diam. of trachea mm.	Diam. of outer cannula mm.	Lumen of inner cannula mm.	Curve of cannula on concave side. Short c.	Curve of cannula on concave side. Long c.	Radius on the concave side mm.
1½–2 yrs. ...	5–6	5.5	4	76 deg.	111 deg.	19
2–4 yrs.	7	6.5	5	81 deg.	101 deg.	21
4–6 yrs.	8	7.5	6	83 deg.	96 deg.	23
6–8 yrs.	9	8.5	7	85 deg.	102 deg.	24
8–12 yrs.	10	9.5	8	82 deg.	103 deg.	25
Adult.	12–23	11–13 and more	9–11 and more	81 deg.	103 deg.	29

The *dangers during the operation* consist in bleeding, which is purely a matter of technic; asphyxia (see above), which usually bows to artificial respiration; and décollement of the mucosa, which is avoided by proper introduction of the tube.

The *after treatment* of the tracheotomized patient, especially in diphtheria cases, calls for constant care, so that the *free passage of air is maintained.* As soon as respiration is embarrassed, the inner tube must be removed and cleansed. The inspired air must be kept moist; this is best accomplished by means of the "croup kettle." DECANNULE-

MENT IS PERMISSIBLE AS SOON AS THE LARYNX PERMITS THE FREE PASSAGE OF AIR.

Decannulement is preceded by testing the patency of the larynx. For the purpose, the tracheotomy tube may be obstructed for increasing periods of time, until the patient sleeps the entire night with the tube occluded; the inner tube may be removed and the extratracheal opening of the outer closed, so that the patient breathes through the window in the latter, or a "weaning tube" may be used. The last consists of an ordinary tracheotomy tube of small size fitted with an obturator. If sufficient air passes the tube with the obturator in place, it may be safely removed.

Decubitus from cannula pressure is a not uncommon sequel of tracheotomy. The pressure ulcer usually corresponds to the point where the lower end of the cannula lies in contact with the anterior wall of the trachea; ulceration of the posterior wall, from pressure by the bow of the tube, is less frequent. Much of this may be prevented by the selection of a cannula suitable to the case (see table). The advent of ulceration is heralded by pain, fever, and bloody expectorate.

Secondary hemorrhage, excluding that from the operative wound — the outcome of injury to blood vessels — is due first to EROSION OF A VESSEL, THE RESULT OF INFECTION, and second to NECROSIS OF THE WALLS OF A VESSEL FROM PRESSURE OF THE CANNULA. A cannula has caused perforation into the innominate vein.

Delay in decannulement is productive of various forms of stenosis.

Granulation stenosis takes its origin in the tracheal wound, usually at is upper angle. The granulations are especially prolific in the larynx. Thost[2] describes a condition of *diffuse granulation stenosis,* in which the entire wound area and the contiguous trachea are invaded, and, in some instances, the cartilage is destroyed. Healing is followed by cicatricial contraction. The condition is ascribed to irritation from the tube and is more marked the higher the wound is located.

The *chief symptom* of granuloma is dyspnea as soon as the tube is removed. The *diagnosis* is made certain by laryngological examination.

The *treatment* of course takes *prophylaxis* into account. This means early decannulement. Removal of the granulation tissue is not easy. In cases of moderate degree, repeated cauterization or curettement accomplishes the purpose. When the wound is closed the latter may be executed by the laryngeal route. *Retracheotomy* is often necessary; this is followed by the more or less prolonged use of suit-

ably fashioned tubes which make even pressure upon the granulations. The carefully adjusted employment of the O'Dwyer intubation method also achieves the desired end.

Deformation stenosis is a term applied to obstruction of the trachea, the outcome of deformation of the tracheal wall by the pressure of the cannula. Figure 831 shows the various forms in which the obstruction occurs and is quite explanatory in this connection. To clarify this still more, one may consider the trachea a semirigid tube susceptible to kinking and to various forms of mutilation, not the least frequent of which is the "incurving" of the edges of the tracheotomy wound. The ultimate outcome of this is stenosis of varying severity. Early decannulement tends to prevent its occurrence. Once established, the treatment is carried out as already stated.

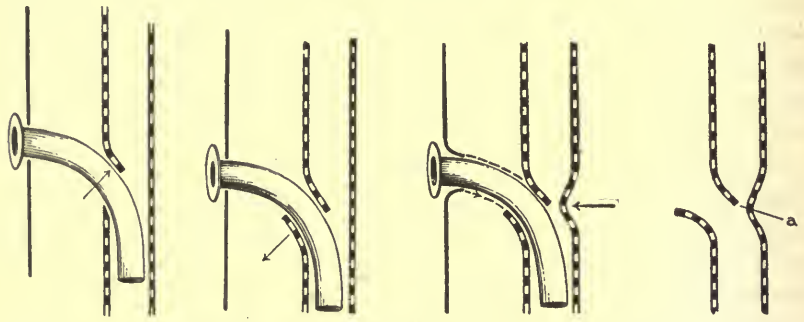

FIG. 831.— POSTTRACHEOTOMIC STENOSES.

Tamponade of the Trachea.— The use of the gauze pack for the purpose of obviating the flow of blood and secretions into the lower air passages during operations in these regions has been replaced by the combination of tampon and cannula. The apparatus consists of an ordinary cannula surrounded with a rubber condom which is distended with air by means of a hand bulb, thus effectually occluding the extracannular portion of the trachea (Trendelenburg[4]). The object may be attained by entwining the cannula with vegetable sponge or other material which swells when it absorbs moisture. Gerster[5] uses an ingenious *umbrella cannula.*

Laryngotomy.— Laryngotomy is essentially utilized for exposure of the interior of the air passages. The larynx may be opened by *partial or complete section* of the thyroid cartilage, or the exposure may include the parts from the hyoid bone to the cricoid cartilage, or the latter may also be divided — total laryngotomy.

The entrance of blood and secretions into the trachea may be obviated by placing the patient in *Rose's position* (p. 774); by the introduction of a *tampon cannula*, or by employing *local anesthesia*, in which event the patient expels collections of fluid through the mouth.

Total laryngotomy (laryngofissure) is a simple operation. The *incision* is made in the median line, extending from the hyoid bone to the cricoid cartilage or a little lower. The sternal muscles are drawn aside, exposing the edge of the larynx. After the conoid ligament and the cricoid ring are divided, the thyroid cartilage is opened directly in the median line in order to avoid injury to the insertions of the vocal cords. A stout knife, scissors, or the Gigli saw may be used for the purpose. The special precautions with regard to the section are taken up in connection with tracheotomy.

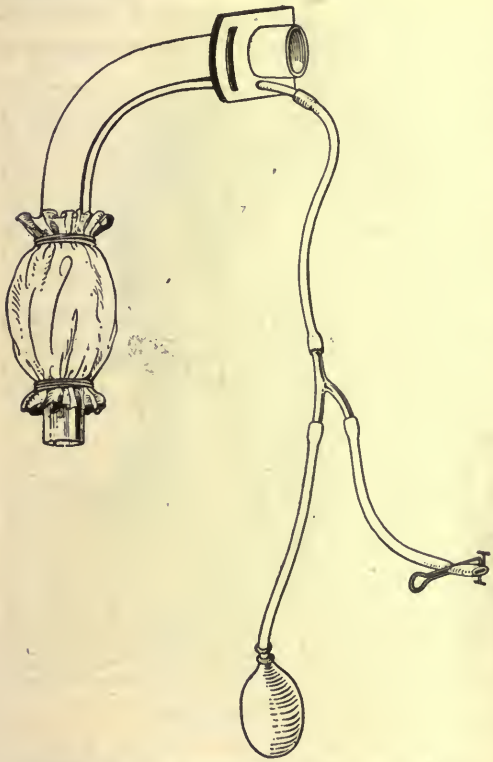

FIG. 832.— TRENDELENBURG TRACHEAL TAMPON CANNULA.

The *after treatment* is aimed at preservation of the phonetic and respiratory functions of the larynx which of course is possible only in accord with the extent of the intralaryngeal operation. The outcome in this connection depends very much upon the care and accuracy with which the sectioned parts are apposed by suture. However, complete closure of the wound should not prejudice the intent of the intralaryngeal technic, nor should tamponade of the larynx be omitted in cases in which this is indicated, especially for the arrest of bleeding. When the operation involves the danger of secondary stenosis, it is customary to suture the larynx over a cannula, which is frequently changed. Gluck[6] has covered the raw surfaces of the interior of the larynx with skin flaps, and v. Bruns uses Thiersch

grafts for the same purpose (see also De Santi,[7] Lejars[8] and Hofmeister[9]).

Laryngectomy.— *Malignant disease* is practically the only *indication for total or partial laryngectomy.* The greater accuracy in diagnosis achieved in recent years has increased the number of successful operations. The technic of partial and complete laryngectomy does not differ to any material extent. The advisability of partial laryngectomy in malignant disease is doubtful. The method of Gluck[6] is largely practiced at this time. While the extent of the operation is great, it must be remembered that this is not objectionable when relief from malignant disease is the intent.

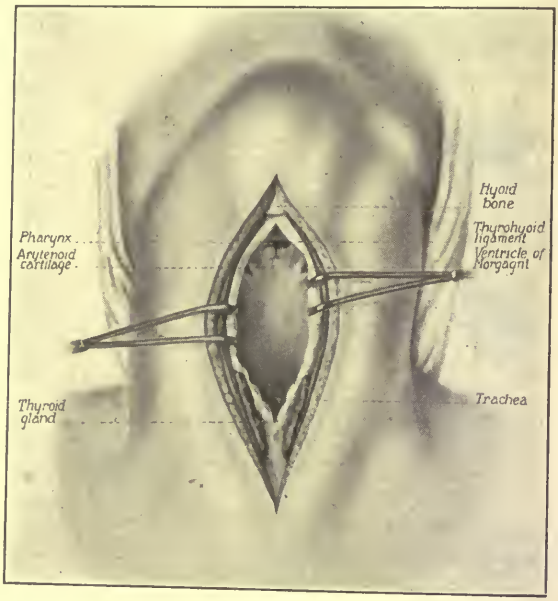

FIG. 833.— LARYNGOFISSURE.

The *skin incisions* are made as follows, one from the hyoid bone to the jugulum and two transverse extending from the anterior border of one of the sternomastoid muscles to the other, the upper being level with the hyoid bone and the lower with the cricoid cartilage. In this way two quadrangular flaps are outlined which are dissected back to their lateral attachments, the superficial veins being tied as the dissection proceeds. Most surgeons simply separate the musculature from the larynx. In the method Gluck[6] follows the sternohyoid, the omohyoid, the sternothyroid and the thyrohyoid muscles are resected, on the ground that they are no longer of use and their removal much increases the accessibility of the larynx, a view with which the writer is inclined to agree. Before doing this, the fascia must be divided to the full extent of the superficial incisions. Next, the isthmus of the thyroid gland is ligated, sectioned, and turned aside, or, if feasible, it

is separated from the trachea and pulled downward. Either of these steps must be patiently carried out. The larynx is now drawn to one side, making the fibers of the constrictor of the pharynx tense; the superior cornu of the larynx is separated from its muscular attachment and the same steps are repeated on the opposite side. The following three arteries are now tied: The anterior division of the inferior thyroid (inferior laryngeal), the cricothyroid and the anterior laryn-

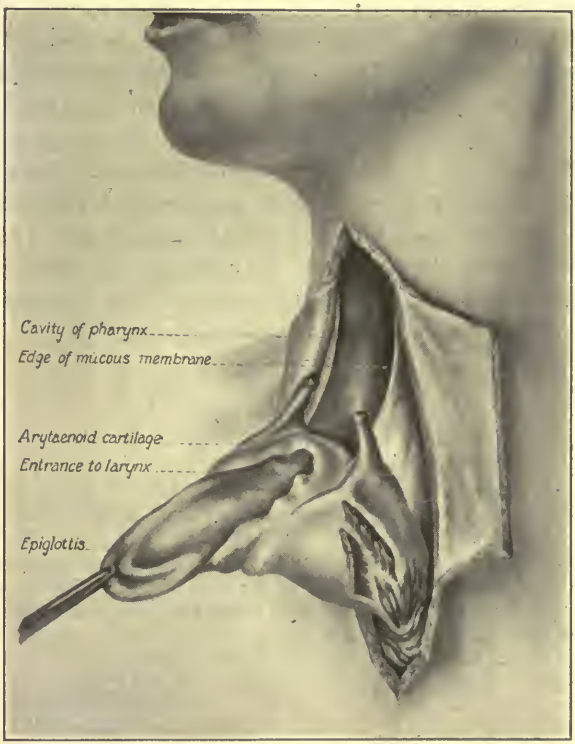

FIG. 834.— COMPLETE LARYNGECTOMY.

geal (branch of the superior thyroid); the laryngeal arteries must be tied before the air passage is opened; the cricothyroid is unimportant and may be tied at any time. The superior laryngeal nerve has no longer any function and may be disregarded.

The larynx is now separated from the hyoid bone by dividing the thyrohyoid ligament close to the hyoid bone, the mucosa is cut just above the epiglottis, and the thyrohyoid membrane is sectioned laterally to the cornua of the thyroid cartilage. The larynx is delivered beyond

the niveau of the wound and is dissected from the pharynx and the esophagus. The patient is still breathing through the larynx, which is not yet invaded, and the narcosis is maintained through this avenue. Finally, a stroke of the knife sections the trachea at the lower edge of the cricoid cartilage and the extirpation is completed. Or, which is better, the defect in the anterior wall of the pharynx is closed with the larynx protruding from the wound. This is easily accomplished with a continuous chromic gut Lembert suture in two layers. The patient still breathes through the larynx by means of an ordinary rubber tube. Now the trachea is sectioned as stated, its stump fastened into the lower edge of the wound and the flaps closed by suture. Drains are introduced through the lower lateral angles of the wound. The *after treatment* is similar to that used in connection with tracheotomy.

The *whispered voice* is remarkably serviceable; however, a certain number of patients find solace in the employment of an artificial larynx. Of these, that of Gluck[6] is perhaps the most useful. The space available here is not sufficient to permit of a satisfactory discussion of this aspect of the situation (see bibliography).

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CHAPTER XI

SURGERY OF THE THYROID GLAND

Goiter.— By far the most frequent disease of the thyroid body is goiter. This may be *endemic, epidemic,* or *sporadic.* Strange to say, it shows a tendency toward geographical distribution. Where goiter is, endemic cretinism is present.

Goiter is rarely congenital, except in the so-called goiter districts. It occurs most frequently in the female sex. This is ascribed to menstruation and pregnancy, special occupations, and the fact that women drink more water than do men. V. Eiselsberg[1] regards the following factors as *predisposing* to goiter; *heredity* and the physiological swelling of the gland occurring during *menstruction, pregnancy,* and *puberty.* It is probable that the character of the drinking water has a causative influence. Perhaps the most extensive investigation with regard to the *cause of goiter* has been made by Bircher, jr.,[2] whose studies of the influence of drinking water seem to have established its etiological connection.

The term *goiter* (*struma*) indicates an ENLARGEMENT OF THE THYROID GLAND irrespective of its character or whether the entire gland or only a portion is affected.

According to v. Eiselsberg,[1] *benign goiters* may be classified as follows:

I. *The diffuse forms.*
   - A. Parenchymatous.
     1. Adenoid with increase of follicles.
     2. Colloid with increase of follicular content.
   - B. Fibrous (very rare) (myxedema?).
   - C. Vascular — associated with active enlargement of blood vessels (aneurismal dilatation) or with passive enlargement of the veins.

II. *The circumscribed forms.*
   - A. Glandular adenoid (local hypertrophy of the thyroid glandular tissue).

B. Colloid with varying metamorphoses; the formation of false and genuine cysts, calcification, ossification, fibrous and hyalin changes of the stroma.

C. Vascular.

D. Adenomata.

1. Fetal adenoma.
2. Tubular adenoma.
3. Papillary cystadenoma.

The *parathyroids* also undergo changes, the most important of which are chronic fibrous parathyroiditis, tuberculosis, cystic and tumorlike changes (Gutknecht,[3] Kloeppel,[4] Landois,[5] Halsted[6]).

Of the *local disturbances* caused by goiter, pressure on contiguous structures is the most important. This includes compression of the trachea, esophagus, the vessels and the nerves.

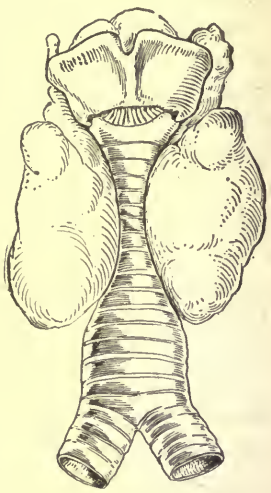

Fig. 835.— Scabbard Trachea.

Pressure on the trachea is evinced by dyspnea and gives rise to displacement, stenosis, or changes in the tracheal wall. A fatal outcome in this connection is not uncommon. The *esophagus* is rarely sufficiently compressed to cause difficulty in deglutition. The *vessels* are uniformly dilated. Interference with the *recurrent laryngeal nerve* causes modification in the voice.

The *symptoms* of goiter are dependent upon the rapidity with which the goiter develops. On the other hand, these are not proportionate to the size of the goiter; a small retropharyngeal nodule, or one located in the thoracic aperture, may give rise to serious disturbances, while a pendulous goiter may hang well down on the thorax without interference with function. The first symptom the patient notices is that the collar is no longer large enough. The dominant symptom is *dyspnea*, especially upon exertion. While this usually becomes gradually more and more marked, if the goiter enlarges slowly the patient may continue to work with but little disturbance in breathing. In a certain number of cases pressure of a comparatively small goiter upon the laryngeal nerves gives rise to spasm and consequent grave interference with respiration (*goiter asthma*). Retrosternal goiters are especially likely to provoke respiratory difficulties. *Circulatory dis-*

turbances are of two kinds, those due to tracheal stenosis and interference with the pulmonary circulation, and the thyrogenous, the outcome of the toxic effect upon *cardiac innervation*. No doubt direct irritation of the vagus and sympathetic nerves in the neck is also a factor in tachycardia. Cyanosis of the face alternating with pallor are the first symptoms in this connection; later, vertigo and syncope

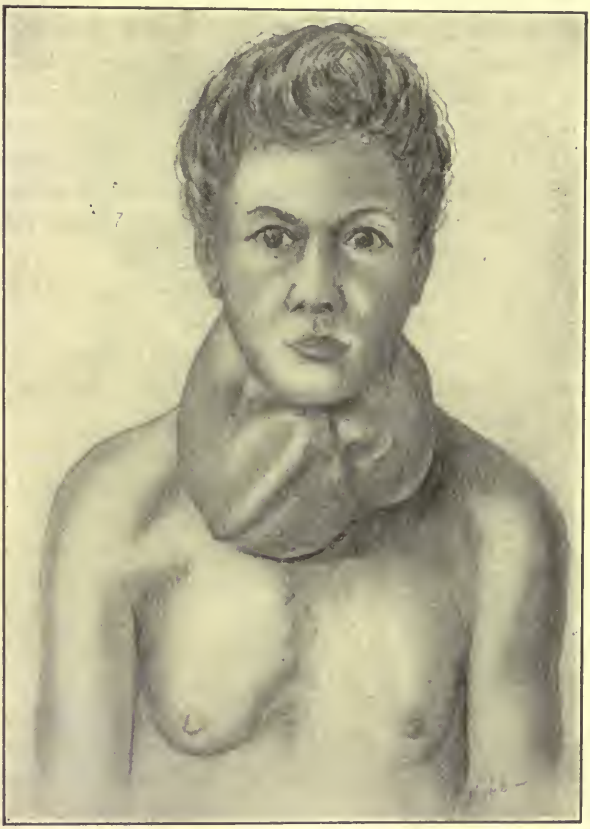

FIG. 836.—COLLOID GOITER, SHOWING DILATED VEINS.

develop and, in a certain number of cases, the heart suddenly dilates and a fatal outcome ensues. The effect of pressure on the recurrent laryngeals is shown by hoarseness, spasm and cough. Prolonged pressure on both nerves results in *aphonia* and may threaten life from paralysis of the muscles of the larynx. Involvement of the trunk of the *vagus* is not common; it is characterized by slowing of respiration.

Pressure on the sympathetic is attended with symptoms of irritation and paralysis. The latter consist in contraction of the pupil, narrowing of the slit of the eyelids, and redness of the ear. Irritation causes dilatation of the pupil.

Abnormal Position of Goiter — Accessory Goiter.— Congenital abnormalities of goiter are variously designated. The term *diving goiter* (goitre plongeant of the French) is applied to a freely movable goiter which moves in and out of the thorax during respiration. *Wander goiter* makes wide excursions, together with the larynx. It may become incarcerated in the thoracic aperture and give rise to severe dyspnea. *Thyroptosis* is a term applied to a goiter in which the entire isthmus and lower poles of the gland are prolapsed into the thorax. A goiter which encircles the trachea and esophagus is called a *ring goiter*, and to those prolonged upward on either side, the term *tubular goiter* is applied.

Accessory goiters are divided into *false and true.* The former are connected with the thyroid gland, the latter are not. The false form is easily diagnosticated, while the true, because of its atypical location, often escapes recognition. Madelung[7] offers the following anatomical classification in this connection:

Glandulae thyroid. access. inferiores arising from the isthmus and extending behind the sternum, and those connected with a lateral lobe and extending into the retroclavicular space (Adjutolo[8]).

Glandulae thyroid. access. posteriores develop from the lateral lobes and extend between the esophagus and the vertebral column. These interfere with deglutition (Krönlein[9]).

Endotracheal accessory goiters are rare. They are due to errors in the embryonic anlage (v. Bruns[10]). See Surgery of the Larynx, p. 1722.

Glandulae thyroid. access. sup. are represented by the pyramidal process type of false accessory goiter which is very common. It often extends to the hyoid bone (see Tracheotomy, p. 1747).

Lingual goiter is of especial importance. It appears at the site of the foramen cecum (Chamisso de Boncourt[11]) in the form of a broadly attached tumor, rarely larger than a hen's egg, usually the size of a cherry (see Surgery of the Tongue, Part IX).

The *diagnosis of struma* may usually be made by examination, irrespective of the history. The examination consists in the employment of inspection, palpation, the laryngoscope, and the Röntgenogram.

Inspection gives information regarding the patient's general con-

dition, such as congestion of the face, dyspnea, sympathetic symptoms (the eye) and also establishes the size, location, and general form of the goiter, and the condition of the superimposed skin. Very often the carotid pulse is visible at the outer border of the sternomastoid muscle. THE FACT THAT THE TUMOR MOVES WITH THE ACT OF DEGLUTI-

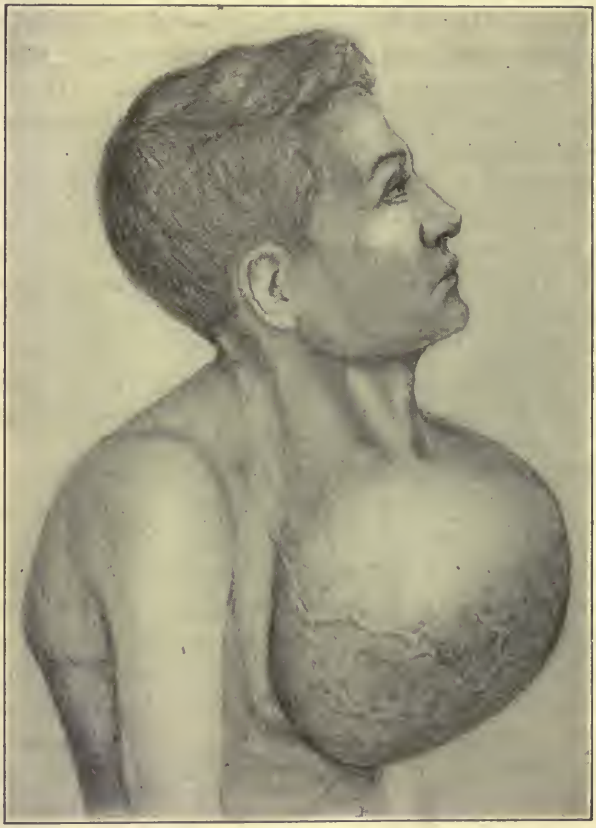

FIG. 837.— ENORMOUS CYSTIC GOITER.

TION IS OF GREAT DIAGNOSTIC SIGNIFICANCE. This symptom is absent in cases of carcinomatous infiltration of the gland.

Palpation reveals the location of the thyroid incisure and of the carotid. Large goiters always push the latter to the outer posterior edge of the sternomastoid muscle. The *consistence* of the goiter must be carefully determined, i. e., whether soft, hard, nodular, or cystic; determination of its motility is equally important.

Auscultation often reveals the presence of a bruit.

It is also important to determine the relationship that the tumor bears to the trachea and larynx. The patient should be examined with the *mirror,* not, as a rule, with the direct laryngoscope; the latter is likely to be very trying. The *Röntgenogram* gives ample information in regard to the degree of tracheal stenosis. *Aspiration* is of doubtful utility. The *therapeutic test,* i. e., the administration of iodin, is helpful in connection with lingual goiter. *The diagnosis should aim at determining the character of goiter.* In this, all the characteristics of the struma weighed against the classification given above must be taken into account.

The Treatment of Goiter.— While the therapy of advanced struma attended with subjective symptoms belongs to the field of surgery, there is no doubt that the majority of those of moderate degree are susceptible to great improvement by internal medication.

The *prophylactic treatment* consists in the avoidance of so-called "goiter districts" or, if this is impracticable, only special drinking water should be partaken of. Under no circumstances must the local water be used for drinking purposes. Filtration does not prevent the deleterious effect of the water.

The Medicinal Treatment.— The administration of iodin was introduced by Coindet (1820). Its efficacy is attested by Kocher,[12] who states that "90 per cent of all goiters are so much improved by the administration of iodin that operative measures are unnecessary." A 10–20 per cent solution of potassium iodid or a 0.1, 1.0, 10.0 per cent iodin ointment is applied to the goiter in conjunction with the administration of 0.1–0.2 gm. of potassium or sodium iodid *pro die.* Saturation of the body with iodin is attended with so-called *iodin marasmus,* the symptom complex of which resembles Basedow's disease. This is best combated with acid sodium phosphate. The treatment is especially efficacious in the hyperplastic and parenchymatous forms of struma; in the fibrous and calcareous forms it is of no avail (Kocher[12]).

Organotherapy owes its efficacy to the iodin content of the gland. It has been much employed in cases of follicular struma (Baumann[13]). V. Eiselsberg[1] does not regard the method as possessing any advantage over the use of iodin. Its objectionable features lie in the tachycardia and vertigo it causes; these may result in a fatal outcome.

The *injection of iodin preparations* has also been abandoned (v. Eiselsberg.[1] See also McCarrison[14] and Crotti[15]).

Operative Technic.— In cases in which the goiter causes menacing symptoms or when the medicinal treatment is not efficacious, operative measures of relief may be employed. These may be divided into (1) *partial extirpation,* in which a greater or lesser portion of the gland is removed; (2) *enucleation* of isolated goiter nodules; and (3) *resection* of a portion of the goiter, which is often a combination of extirpation and enucleation.

With regard to *anesthesia,* one may say that experienced goiter

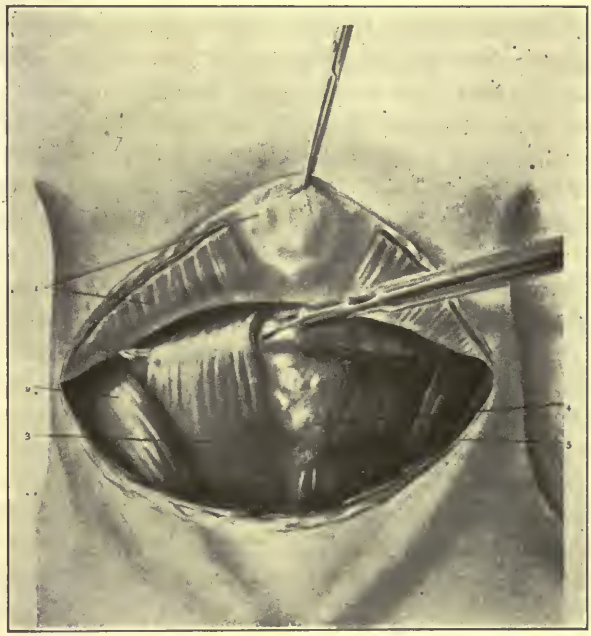

FIG. 838.— EXTIRPATION OF GOITER (Kocher).

1. Skin flap with fascia and platysma. 2. Sternomastoid muscle. 3. Sternohyoid muscle. 4. Goiter. 5. Sternothyroid muscle.

operators use local anesthesia, but that carefully administered ether narcosis does not increase the dangers of the operation.

The *incision* of approach may vary; however, as a rule, the transverse section of Kocher[12] (kragenschnit) is universally employed, except for removal of small nodules when an atypical incision may be used. The *transverse incision* has a slight downward convexity from one sternomastoid muscle to the other and is centered over the dome of the goiter (Figure 838). The median veins of the neck are tied.

The muscles are divided as shown, including the omohyoids; the muscles are subsequently sutured. The goiter is usually spontaneously luxated beyond the niveau of the wound. The vascular middle fascia is divided, exposing the capsule of the struma.

In *partial extirpation* the goiter sound or the finger delivers the upper pole beyond the skin (this must not kink the trachea) and the superior thyroid vessels are tied and cut; the lower pole is next delivered and the inferior thyroid vessels are ligated and divided between the ligatures. Care is taken not to traumatize the *parathyroid bodies*. The recurrent laryngeal nerves are best avoided by leaving the posterior portion of the gland *in situ*. The greater part of both lobes, or one lobe in its entirety, may now be removed. Unilateral strumectomy is the method of choice (Kocher[12]). The isthmus may be ligated or crushed with forceps and sutured. *Bleeding must be carefully arrested.* A drain (rubber tissue) is passed through the wound. Halsted[6] closes the superficial wound with a subcuticular suture.

Enucleation is the outcome of a desire to avoid injury to the nerves, to be sure that enough gland is left *in situ*, and to avoid trauma to or removal of the parathyroid bodies. The technic of exposure is similar to that already described; the nodule or nodules are located by palpation and the afferent vessels are encircled by transfixion sutures and tied. The parenchyma is then incised and the doomed structures enucleated with the goiter sound or the finger. Oozing is controlled by packing and ultimately drained with gauze. The method is now restricted to the *enucleation of cysts* (Kocher[12]).

The combination of *extirpation with resection* and *extirpation with enucleation* is a modification of v. Mikulicz's method introduced by himself. After the superior thyroid vessels are tied, a wedge-shaped portion of the gland on either side, extending down to the isthmus, is excised and the resultant wound is sutured. Its technic and its bearing on preservation of the parathyroid bodies has been carefully worked out by Pool and Falk[16].

Recurrences after goiter operations are regarded in the sense of compensatory hypertrophies. Clinically, they may be divided into those that do and those that do not cause disturbances (other than cosmetic effect). Of 600 goiter operations performed in v. Bruns[10] clinic, only twelve required secondary operation. The symptomatology of recurrent struma is similar to that of the primary affection.

The Dangers of Goiter Operations.— The operative fatalities of

goiter operations in Reverdin's statistics, comprising 6103 cases, show a mortality of 2.88 per cent; Kocher's mortality of recent years is 0.2 per cent (Crotti[15]). Of the complications, *bleeding* is the most serious; the danger of *air embolism* is much exaggerated. Of the nerves, division or injury of the *recurrent laryngeal* is a distressing occurrence. *Infection* is an exceedingly menacing complication. Fortunately it is rare and is obviated by care in the execution of asepsis.

The special dangers of struma operations may be separated into two groups, *tetany and cachexia.*

Tetania parathyropriva is comparatively rare, as total extirpation of the thyroid gland is no longer practiced and thus the parathyroid bodies are spared.

The disease is characterized by convulsive seizures — resembling tetanus — which attack the muscles of the extremities, the larynx and the diaphragm; the last may prove fatal. Tetany may begin immediately after the patient emerges from narcosis or several days (up to ten) later. As a rule, a prodromal period is observable, a feeling of discomfort, stiffness and weakness of the general musculature — especially in the muscles of the forearm and calf. The *diagnosis* may often be based upon these symptoms, together with the demonstration of Chvostek's or Trousseau's signs. In the former, a sharp tap over the facial nerve in the parotid gland provokes spasm of the facial muscles on that side; the second consists of a cramp of an extremity when pressure is made on its main artery or nerve.

In *cases of moderate degree* the disturbances are limited to tonic spasm of the upper extremities, especially the forearm; the elbow, the wrist and the basal phalanges are flexed, the other finger joints are extended; the thumb is apposed to the palm, that is, the typical "obstetrical hand."

In *severe cases* the backs of the hands are turned toward one another (in the manner seen in "siren malformation"), the lower extremities are contracted, the hips and knees are extended, the feet and toes are held in plantar flexion. Contraction of the muscles causes pain, the pulse is rapid, fever is absent; the sensorium remains clear (unfortunately).

This picture is paroxysmal, the attacks varying from two to fifteen minutes in duration and extending over several days at a time. Death is the outcome of diaphragmatic fixation. A certain number of cases recover spontaneously, while not a few go on to *chronic tetany.* Tetania parathyroidopriva has followed partial strumectomy; this is

explained on the ground that the epithelial bodies vary widely as to location and may thus be inadvertently injured or removed, despite the employment of a technic designed to avoid them (see Nos. 14, 15 and 16 of bibliography). The affliction has followed ligature of the four thyroid arteries (v. Eiselsberg[1]).

Spontaneous improvement of tetany has occurred in accord with demonstrable regeneration of thyroid tissue, which probably included growth of parathyroid substance. In this connection, v. Eiselsberg[1] reports an instructive case. Of 1157 cases operated upon in eleven years in the clinic of this persistent worker, thirteen developed tetany of which three died, seven of the remaining ten gradually recovered, and three slowly improved after years of invalidism. One of these was ultimately benefited after two transplantations of thyroid bodies. The first attempt was not followed by much improvement; the second time, a fresh parathyroid was transplanted from a suicide and was followed by marked lessening of the symptoms.

Cases of *moderate postoperative tetany,* in which a slight *Chvostek* is present, seem to be benefited by the administration of calcium lactate (6–9 gms. p. d.). Halsted[6] reports favorable results following the administration of parathyroidin derived from the sheep. In *menacing cases,* transplantation of parathyroid bodies comes under consideration (Pool-Falk[16]).

Cachexia thyroprivia develops slowly after strumectomy and may be secondary to tetany. *Myxedema operativum chronicum* was first described by Reverdin.[17] The dominant characteristics of the disease consist in lessening of mental capacity, intelligence and energy and in an edematous swelling of the skin. In young persons general bodily development is inhibited.

Many patients remain normal for a long time (years) after the operation and then the symptoms stated above gradually develop. The changes in the skin make the diagnosis patent. The skin of the face is dry, waxy and "puffed" and the excretion of sweat and the secretion of sebum are arrested. The hair loses its luster and may fall. The lower lids sag, the play of expression is absent and the features convey the notion of apathy. The edema, which does not pit, slowly extends to the extremities and trunk. In the supraclavicular regions it protrudes in a form bearing some resemblance to lipoma. In not a few instances the mucosa of the mouth is affected, so that speech is thick and indistinct — a distressing accentuation of the idiotic picture. The blood shows domination of hemoglobin content

and lessening of leukocytes. In children, the long bones cease to grow (v. Bruns[10]), while the diameter of the epiphyses continues to increase. Sexual development is inhibited or remains stationary. The patient's resistance to cold is decreased.

According to the collected statistics of v. Eiselsberg,[1] 70 per cent of the cases subjected to total strumectomy show acute or chronic myx-

FIG. 839.— CACHEXIA THYROPRIVA (Myxedema).

edema and tetany. Of 900 cases of partial strumectomy, only one (for malignant disease) showed cachexia (Kocher[18]). Of 2000 cases operated upon by the Mayos,[19] 2 developed cachexia.

The observation that cachexia is prevented by leaving sufficient glandular structure *in situ*, and that when omission to make proper provision of this sort has been followed by cachexia this disappeared as the residual glandular tissue grew, led to the development of an

adequate *therapy* in this connection. In these cases, organotherapy, in the form of implantation, injection and administration of the thyroid tissue, has achieved a notable triumph. At present, the administration of thyroid extract is universally practiced.

Ligature of the Thyroid Arteries.— Ligature of the thyroid arteries (Wölfler[20]) is practiced for the relief of *parenchymatous struma,* especially when this form of goiter appears in young persons and grows rapidly. To accomplish the purpose, all of the four arteries must be tied at one sitting. Cachexia and tetany has followed the measure. The method has a distinct field of usefulness in Basedow's disease.

Luxation of the Goiter.— Exothyropexia (Poncet et Jaboulay[21]) consists in exposing the goiter as stated above and permitting it to prolapse through the wound. Gradual atrophy of the gland takes place. In cases of goiter attended with great pressure, the procedure may save life.

Tracheotomy in Connection with Goiter.— Tracheotomy in connection with goiter is a palliative measure. The masked position of the trachea makes this an extremely difficult procedure (see also Berry,[22] Hastings[23] and Verebely[24]).

Inflammations of the Normal Thyroid Gland and of Goiter.— The similarity of the inflammatory processes invading the thyroid body and goiter justifies considering them together. Of the various forms of goiter, the follicular and cystic are most often affected.

Thyroiditis is usually the result of *direct injury* with subsequent infection; or it occurs in connection with a general infection and is then of metastatic origin. *Metastatic thyroiditis* or *strumitis* develops in the course of typhoid, pyemia, puerperal fever, pneumonitis, scarlatina, variola, diphtheria, and rheumatism. Acute suppurative strumitis has been caused by infection with the bacterium communis coli (Brunner[25]).

The *symptoms* are those common to all infectious inflammatory processes. Rapid swelling of the gland may provoke respiratory disturbances. The process results in *resolution* (rare), *suppuration, and necrosis. Suppuration* may perforate externally or into the trachea or esophagus; erosion into the carotid artery has occurred. *Necrosis* is simply a severe form of inflammation.

The *treatment* is similar to that of all inflammatory processes. *Incision must be liberal and not too long delayed.* V. Eiselsberg[1] has observed fatal tetany in connection with severe, acute, purulent thy-

roiditis. Autopsy indicated that the parathyroids had been destroyed. A residual fistula calls for partial thyroidectomy. A low grade of strumitis may simulate malignant disease.

Specific Thyroiditis.— Tuberculous thyroiditis appears in two forms. The one form consists in *miliary nodules* and is a part of general miliary tuberculosis. The second, which is rare, appears in the form of *tuberculous nodules* which give rise to pain and dyspnea. Involvement of the contiguous lymph nodes gives the process the appearance of malignant disease. As a rule, the microscopical findings clear up the diagnosis (Claremont[26]).

Actinomycosis of the thyroid gland is very rare. It has been responsible for myxedema (Köhler[27]).

Lues of the thyroid gland occurs in the secondary stage of the disease as a part of the lymphatic disturbances; it is transient in character. In the *tertiary stage,* diffuse luetic thyroiditis may be responsible for myxedema (Küttner[28]). However, the gummatous form of the disease possesses the greatest surgical importance. Like tuberculosis, it presents a picture very similar to that of malignant disease. The *diagnosis* would have to rest on the therapeutic test, together with the serological findings.

Echinococcus of the thyroid gland presents itself with the clinical manifestations of a cystic goiter. A cystic goiter attended with "purring" and urticaria may suggest the diagnosis. This may be verified by aspiration and the serum reaction (p. 1325). Enucleation of the process is the therapy of choice (Vitrac,[29] Ultzmann[30]).

Tumors of the Thyroid Gland.— Aside from adenomata (alluded to above) benign tumors of the thyroid gland are very rare. This consideration has some diagnostic significance with regard to the specific infections and malignant disease. *Carcinomata and sarcomata* occur most often in goitrous thyroid glands. In 7,700 autopsies, thyroid carcinoma was found eleven times and sarcoma five (Chiari[31]).

Sarcoma of the thyroid appears in the form of a rapidly growing tumor beginning in a lobe and soon involving the entire organ. The growth soon perforates the capsule and invades the various structures of the neck. Operative removal holds out the possibility of relief only when performed early in the disease. *Carcinoma* makes its initial appearance in the form of a circumscribed infiltrate or nodule. As a rule, the entire gland is soon invaded, although adenocarcinoma and scirrhus may remain restricted to a single lobe. The capsule is perforated early in the disease; this is soon followed by difficulties in

swallowing and deglutition. *Metastases* are very common. Of the various forms of carcinoma, the *alveolar* is most frequently found.

At first, the *diagnosis* is difficult; later, it is unmistakable. *When, in an elderly person, a goiter which has persisted for a long time suddenly increases in size, and at the same time the gland becomes hard, the lymph nodes enlarge and lancinating pain appears, together with dyspnea and dysphagia, the diagnosis of cancer may be made with reasonable certainty.*

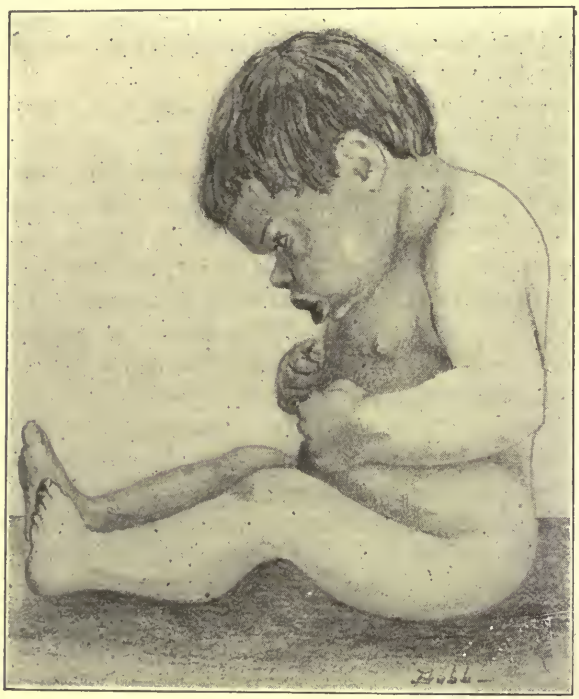

FIG. 840.— CRETINISM (Eleven year old child).

Operative removal is indicated only when the capsule is not perforated. An effort should be made to spare the parathyroid bodies (see Nos. 14, 15 of the bibliography).

Excision of a specimen for microscopical examination in suspected malignant disease is justifiable only if immediate operative removal is contemplated. The opening thus made in the capsule favors rapid extension of the disease to the neighboring tissues (v. Eiselsberg[1]).

Cretinism.— Goiter is an early symptom of the degenerating processes, the last grade of which is cretinism. Bircher[2] holds that the

two processes simply occur together. Studies of the affection in the Jewish race seem to show that a cretinic generation follows one of goiterism (Flinker[32]). The causative influence of the drinking water of certain districts has been demonstrated by v. Wagner.[33] Cretinism is characterized by (1) disturbances of growth, specific changes in the skin, and incomplete development of the genitals; (2) idiocy; (3) goitrous degeneration, or absence of the thyroid gland.

The arrest of growth and the changes in the skin have already been discussed. The abnormalities of the genitals in cretinism are especially characteristic. The penis, scrotum, labia, and uterus remain in a prepubertal condition. However, the capacity of reproduction may appear later in life, so that a cretinic woman may bear children. *Idiocy* is regarded as due to underdevelopment of the brain (v. Wagner[33]); hearing is the most seriously impaired of the special senses (Kocher[12]). The thyroid gland is always abnormal; the parathyroids seem to be unaffected (Chiari[31]). The administration of thyroid tissue is followed by improvement. This, in some instances, is startling (v. Eiselsberg[1]).

Spontaneous myxedema in the adult occurs when the function of the thyroid gland is lessened. *It is attended with changes in the skin and in the psychic attitude of the patient.* It is too late for disturbances in development to appear. The changes in the skin are similar to those already discussed. The *psychic disturbances* consist in apathy and stupidity. Charcot compares the mental condition to that of a hibernating animal. Speech is impaired "as though the speech mechanism were frozen" (v. Eiselsberg[1]). The *thyroid gland is either goitrous or decreased in size;* the latter is the rule. The notion that hypothyroidism is responsible for the clinical picture of myxedema is supported by the analogy it bears to cretinism. The disease is insidious in its onset and may escape recognition in its early stages. The administration of thyroid preparations, especially of Baumann's[18] thyroiodin, is of signal service.

Exophthalmic Goiter (Basedow's, or Graves', Disease).— The *dominant symptoms* of Basedow's disease consist in *struma, exophthalmos, tachycardia and tremor,* to which may be added a number of nervous disturbances. As a rule, the thyroid gland is enlarged, tender, hard, uniformly granular (like the mammary gland of a woman during lactation — Kocher), and very vascular. A bruit may very often be heard over the thyroid arteries — before the gland enlarges; however,

struma usually is not marked. Section shows an almost complete absence of colloid.

The histological findings may be stated as follows: (1) Epithelial proliferation with the production of larger and smaller hollow spaces and tubules, canals, and papillary excrescences. (2) The epithelial cells vary in size and form, being cylindrical in the tubules, and show a tendency to desquamate. The epithelial garlands are irregularly formed. (3) Most of the sectors of the gland are sparingly furnished with typical colloid; the spaces are filled by cysts and tubules, which contain a pale, granular, rarely homogenous material. The striped and unstriped muscular fibers are degenerated.

Fig. 841.—Exophthalmic Goiter (Falk).

Exophthalmos is attended with additional eye symptoms as follows: (1) *V. Graefe's sign*—the upper eyelid does not follow the eyeball in its vertical excursions; (2) *Stillwagon's symptom*—abnormal separation of the eyelids. The upper eyelid is permanently retracted. *Möbius' symptom*—insufficient convergence of the eyeball without diplopia.

Tachycardia is an early symptom; the pulse often reaches 120–140 per minute. A coexisting myxocarditis usually results fatally.

The *blood picture* is characteristic, leukopenia with diminution of the neutrophiles and increase in lymphocytes. The coagulation period of the blood is increased.

The *tremor* is like that of alcoholism, but, unlike this, is not limited to the extremities.

The *skin* shows evidence of vasomotosecretory disturbances—increased diaphoresis, abnormal pigmentation, edema and insular hyperemia. Attacks of diarrhea, emaciation, muscular weakness, and hysteria are common occurrences.

The administration of thyroid substance always increases or provokes tachycardia.

The *cause* of Graves' disease was for a long time believed to lie in disorders of the nervous system, especially the sympatheticus and the medulla. It is certain that this notion is not susceptible of scientific proof. The theory of the day is that of Moebius, who ascribes the disease to an OVERPRODUCTION OF, OR AN ABNORMAL CHANGE IN, THE CHARACTER OF THE THYROID SECRETION, i. e., hyperthyroidism, or perhaps dysthyroidization. The fact that the symptoms are so exactly contrary to those produced by hypothyroidism makes the theory sound rational.

The disease appears most frequently in women at or near puberty. As a rule, it runs a chronic course. Spontaneous healing is rare, although remissions are frequent; palliative treatment is illusionary.

The *diagnosis* in defined cases is easy. Early in the disease, and in mild cases, the affections which provoke one or more of the symptoms must be excluded.

The *treatment* of Basedow's disease by medicinal agents, including Moebius' serum, need not be discussed here. The Röntgen ray treatment is perhaps of value. Its employment makes subsequent operative treatment more difficult. While not every person presenting symptoms indicative of hyperthyroidism need be subjected to an operative procedure, there is no doubt that no other method of treatment achieves the same results. The various operative methods, including ligature of the arteries, show an average mortality of about two per cent. In this connection the statistics of the Mayos are of interest. These show 70 per cent cured and the rest improved. At this writing partial thyroidectomy is the operation of choice. The ligature of the arteries should be restricted to cases in which the more extensive operations are manifestly dangerous. This viewpoint seems to be that of Kocher, the Mayos, and v. Eiselsberg. Kocher and v. Eiselsberg use local anesthesia; the Mayos and Crile employ general narcosis. The latter is particularly careful not to expose his patient unnecessarily to the "horrors of operation," a conception one cannot help but

give serious consideration, especially when the nervous symptoms coincident to the disease are taken into account. On the other hand, local anesthesia, in the hands of the skilled technician, applied to patients to whom the necessary manipulations are not a "horror" possesses many advantages. The persistence of the thymus does not constitute a contraindication to the operation, but entails special care in its execution. *Exothyropexia* is no longer practiced for the relief of hyperthyroidism, nor is *resection of the sympathetic in the neck* regarded as serving a useful purpose. *Thymoidectomy* for the relief of the symptoms of hyperthyroidism is not yet an established procedure.

A *recapitulation* of the clinical manifestations attendant upon diseases of the thyroid gland and the parathyroid bodies may be arranged as follows:

A. Hypothyroidism.
1. Spontaneous hypothyroidism.
   a. Endemic, congenital and acquired cretinism.
   b. Sporadic cretinism, infantile myxedema.
   c. Spontaneous myxedema of the adult.
2. Traumatic hypothyroidism (cachexia thyropriva).

B. Hypoparathyroidism.
1. Spontaneous tetany (in children).
2. Postoperative tetania parathyropriva. Transition from spontaneous into traumatic tetany occurs in suppurative or other destructive lesions of the parathyroid bodies.

C. Hyperthyroidism (morbus Basedowii).

D. Hyperparathyroidism, not yet understood but responsible for certain nervous disturbances, such as myasthenia gravis and paralysis agitans.

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The literature of exophthalmic goiter is too extensive to be included here. The reader is referred to Nos. 14, 15 of the bibliography and to Kocher's "Le prix Nobel."

PART XIII

THE SURGERY OF THE THYMUS GLAND

CHAPTER I

DISEASES OF THE THYMUS GLAND

Thymus Hyperplasia.— Qualitative changes in the parenchyma of its glandular structure is the basis of hyperplasia of the thymus. Usually, the medullary portion is increased and the capsule atrophied. The hassall corpuscles are diminished in number, enlarged and present regressive changes. Simultaneous hyperplasia of medulla and capsule is rare.

On the one hand, thymus hyperplasia is expressed in the form of a congenital insufficiency; on the other, it is attended with evidence of either dysthymization or hyperthymization. The process is always a part of more or less extensive changes in the entire lymphatic apparatus. The occurrence of sudden death in cases of demonstrable thymus hyperplasia is ascribed to an abnormal constitutional lymphaticochlorotic dyscrasia which affects the intracardial centers and results in arrest of cardiac function. This may happen after even slight external irritation. While this explanation accounts for a certain number of "*narcosis deaths,*" it has also been shown that in not a few instances, the fatal outcome in children is due to suffocation and that dysthymization only plays a secondary part (Hammar[1]). Klose[2] seems to have shown that the irritation of surgical attack upon the gland is followed by regeneration of healthy thymus tissue. This accounts for the local relief and the beneficial constitutional effects which follow the restriction of the abnormal internal secretion of the organ. Complete restitution to the normal is not to be expected, on the ground that the underlying causative factor is not removed by the surgical measures (see also Crotti[3]).

In the *causation* of thymus hyperplasia and aplasia, *heredity* plays an important part. The influence of *chronic alcoholism* in the parentage is undoubtedly the most important factor in this connection. The causative relationship of lues is also clearly established. It is probable that the influence of consanguinity has been overestimated. Disturbances of respiration constitute the chief indication for operation.

Thymic stenosis develops in the first year of life. The infant breathes noisily; this is especially marked during excitement and persists night and day. Respiration is attended with a snoring, bleating or gurgling sound. The epiclavicular depressions are exaggerated during inspiration. Infants with thymic stridor are always affected with hyperplasia of the thymus. Persistent dyspnea is often exacer-

FIG. 842.— RÖNTGENOGRAM OF AN ENLARGED THYMUS (Harlem Hospital case).

bated and occasionally one of the attacks proves fatal. The attacks are precipitated by any posture or mechanical influence which produces venous stasis in the neck.

The *diagnosis* is based on the *physical* and *biological* findings. *Percussion* reveals the dimensions of the gland, which throws a clearly defined shadow upon the *Röntgenographic negative*. The *blood* shows a marked *leukocytosis* (80 per cent). Thymic stenosis should be recognized by the respiratory embarrassment. *Esophageal stenosis*

is not as common as the tracheal. Difficulty in deglutition is an indication for operation, especially if tracheal stenosis is also present — which is usually the case. Dyspnea without a lesion of the nose or throat should lead to the suspicion of thymic stenosis. The *vagus* is often included in the pressure zone of a hyperplastic thymus gland, but is probably not directly responsible for a fatal outcome. Slowing of the pulse rate indicates vagus irritation. Pressure on the blood vessels is a distinct indication for operation.

Narcosis death in children may often be ascribed to pressure caused by thymus hyperplasia. The mechanical causation does not, however, explain the *fatal outcome in the adult*. This would seem to be properly ascribed to cardiac shock. In these cases, *hyperplasia of the thymus* and disturbances in the chromatic system (hypoplasia) are regarded as the causative factor. It has been shown that the thymus gland furnishes a hormone which is antagonistic to the action of adrenalin upon the vagus (Wiesel,[4] Marfan[5]).

Circulatory and Inflammatory Disturbances of the Thymus Gland.— A normal thymus gland may become secondarily enlarged and cause death by pressure. Congestions, hemorrhages, and necroses have been observed in connection with inflammatory processes in the neck, especially those occurring in the course of infectious diseases (diphtheria). Diffuse hematomata occur as a part of *melena neonatorum*. Obstetricians call attention to changes in the thymus resulting from trauma during delivery of the infant. The lesion is one of multiple hemorrhagic areas of infiltration. No doubt the condition is often responsible for asphyxia in the newly born. Suspicion of pressure from this cause justifies decompressive incision of the gland in the jugulum.

Metastatic abscesses occur in the exanthema of children. Isolated *gumma* and solitary *tubercle* are rare. *Leukemic* and *granulomatous* infiltrations develop in connection with similar processes in the mediastinal lymph nodes.

Tumors of the Thymus Gland.— New growths of the thymus present themselves in the guise of slowly growing tumors of the mediastinum. The *diagnosis* is not made until the affection is sufficiently advanced to cause pressure, and then it must take a number of conditions into account. The Röntgenogram is of particular diagnostic value.

Of the *benign tumors, lipomata, dermoids, and ciliated epithelial cysts* are the most common.

Sarcoma is more often found than is carcinoma. The former has been responsible for the clinical picture of *myasthenia gravis pseudo-*

paralytica (Hammar[1]). *Carcinoma* is usually of the medullary form.

Thymus Gland and Graves' Disease.— Although isolated diseases of the thymus gland may produce symptoms of constitutional poisoning resembling Graves' disease, clinical experience does not give us the right to characterize the process as "thymogenous" Graves' disease. The part that the thymus gland plays in the clinical picture of Graves' disease has been recently established. The special symptoms for which it is responsible are blood changes, i. e., *lymphocytosis and toxemia* (Klose[2]).

The pathological changes in Graves' disease are not influenced by excision of the thyroid gland. If these are to be remedied the operative attack must include resection of the thymus with that made upon the thyroid gland. However, this indication is only relative, as experience teaches that the symptom complex of Graves' disease disappears entirely as the outcome of the thyroid operation, i. e., return to the normal, occurs without correction of the blood picture (Klose[6]).

The influence that the toxic components of a qualitatively altered thymus gland have upon the symptom complex of Graves' disease may be established only to a presumptive degree. No doubt the *vagotonia* observed in certain cases is one of these symptoms. It is also probable that the degree of *lymphocytosis* in Graves' disease is not indicative of the severity of the disease, nor does it mean that the thymus gland is playing an increasing part in it. This is none the less so because lymphocytosis of itself very properly may be regarded as a condition of affairs prejudicial to the organism. However, it may be considered as certain that the degree of lymphocytosis in Graves' disease points to dysthmization and justifies SURGICAL ATTACK UPON THE THYMUS GLAND WHEN A RELATIVELY SLIGHT INCREASE IN THE SIZE OF THE THYROID GLAND IS ATTENDED WITH SEVERE CARDIAC DISTURBANCES. A faulty conclusion in this connection may be arrived at when previous Röntgen ray treatment has caused lessening of lymphocytosis. The ray treatment may cause an involution of the thymus gland from which regeneration does not occur. It would seem best to employ the measure for the purpose of postoperative reduction of lymphocytosis in Graves' disease.

Operations on the Thymus Gland.— The surgical treatment of thymus disease aims at decompression and restriction of its secretion. These are attempted in the hyperplasia of childhood. In Graves' disease the treatment is directed only toward excluding abnormal secretion.

In childhood only a portion of the gland may be removed; in the

adult, it is permissible to extirpate the entire organ. In the adult *radiotherapy* may accomplish the purpose; in childhood its employment is absolutely contraindicated — the persistent application of the method by some notwithstanding.

All forms of pressure are relieved when the upper pole of the gland is resected and its capsule fastened to the sternal fascia. This reduces the dimensions of the organ and lifts it from the mediastinum.

Intracapsular excision and fixation of the thymus gland accomplishes the purpose in most instances. When the gland is particularly large, the *manubrium may be resected.* This permits the gland to protrude and relieves pressure to a sufficient extent, without dangerous reduction of its tissue. In very severe cases it is justifiable to perform

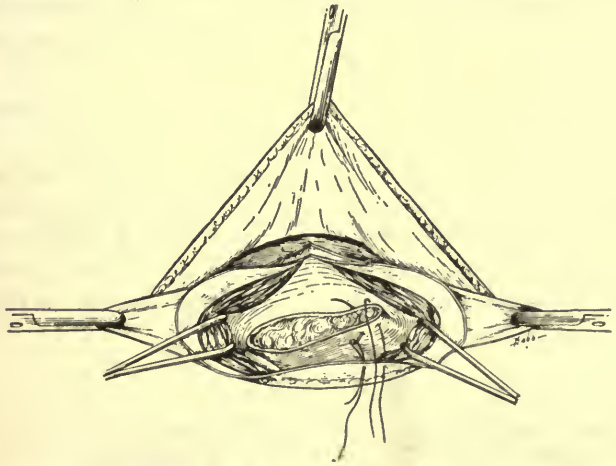

FIG. 843.— CERVICAL SUSPENSION OF THYMUS GLAND (Klose).

intracapsular enucleation of one of the lobes of the gland. As the left lobe is the one most often affected, lies higher, and makes pressure on the vagus, it is the one to be sacrificed. This leaves the larger, right lobe to perform the necessary functions.

Ether narcosis is desirable. An incision about six cm. in length, with a slight upward concavity, is made over the lower portion of the incisura jugularis, through the skin, and the flap turned upward. The superficial fascia is opened in a vertical direction. The sternal muscles are separated, or, if necessary, incised transversely. The fascia lying behind these muscles is opened, also in a vertical direction, after the veins are carefully tied. This exposes the pretracheal space, in which either the pointed ends of both lobes, or only that of the left,

covered with the capsule, are found. As the latter is grasped and drawn forward, it is seen to be continuous with the sheaths of the carotid arteries and jugular veins. In children it is impossible to extirpate the entire gland.

As the capsule is opened the parenchyma of the gland promptly prolapses into the wound. This is often immediately followed by abolition of stridor, and suffices to remove the extruded tissue. If the stridor persists, enough of the gland is delivered until this ceases. The anterior layer of the gland is sutured to the sternal fascia and the wound tamponed.

Resection of the sternum is executed in accord with general surgical principles. The prognosis is favorable. A similar technic is followed in cases of Graves' disease. EVERY STRUMECTOMY SHOULD BE PRECEDED BY PALPATION OF THE THYMUS AND THIS, WHEN ENLARGED, RESECTED (Klose[6]).

When the thymus gland is not movable, it must be approached by *resecting the sternum.*

When the gland "enmantles" the heart, operative attack must be performed with proper consideration for differences in pressure (see Surgery of the Thorax, Part XV, chap. xvi). A similar method is used in removal of tumors of the thymus gland. If this is not feasible, the pressure symptoms may be relieved by *median section of the sternum.*

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PART XIV

THE SURGERY OF THE ESOPHAGUS

CHAPTER I

THE SURGERY OF THE ESOPHAGUS

Examination of the Esophagus.— Esophagoscopy has had a sharp impetus in the last few years. In March, 1919, Jackson[1] analyzed 628 cases of foreign body removed from the air and food passages. Unfortunately, it has been practiced more by the laryngologist than by the surgeon. It is impossible to review the history of the development of this branch of surgery here. It had its origin in the work of Mikulicz and passed through stages of improvement to the development of the present method of electrobronchoesophagoscopy of Brunning (see Part XII, chap. v). As a rule, the application of novocain to the pharynx (especially the sinus pyriformis) is sufficient to make the introduction of the instrument bearable; in special cases, narcosis may be employed. Usually, the tube is introduced with the patient in the sitting posture. When the constrictors of the pharynx are passed, the patient is carefully lowered to the supine position. The subsequent advancement of the tube is now easily accomplished. If the instrument is halted, the cause must be sought for. When the instrument is introduced without the mandarin (which is indicated in lesions located high in the esophagus), especial care must be taken not to do injury. Recently Aeh[2] has devised an instrument much like the proctoscope, which makes possible inflation of the esophagus. It is used in retrograde or gastral esophagoscopy and may also be used through an esophageal fistula in the neck. It has recently been employed in oral examinations and would seem to point the way to a field of usefulness of considerable magnitude.

The employment of esophagoscopy is contraindicated in acute inflammatory processes (except for foreign bodies), emphysema, cardiac diseases, aneurism and tracheal stenosis. ESOPHAGOSCOPY IS NOT AN ENTIRELY HARMLESS PROCEDURE, but the introduction of the bougie is little less so. Infection of the esophageal mucosa, phlegmon, and invasion of the mediastinum has led to a fatal outcome in several instances. This means that all manipulations in the esophagus must be

carefully executed. The entrance of the esophagus, especially in cases of carcinoma, presents the zone of greatest danger in this connection.

The field of usefulness of the method includes the removal of and localization of foreign bodies, and has a *diagnostic* and *therapeutic value.*

The *diagnostic value* pertains to visualizing various lesions, such as new growths, ulcers, strictures, dilatations, etc., and their localization. In addition to this, portions of tissue may be removed for microscopical examination. *Therapeutically,* the method permits of the local treatment of dilatations, strictures, etc., and the opening of abscesses, etc.

Radioscopy is of great diagnostic assistance. See Foreign Bodies, Strictures, etc.

Auscultation reveals two "deglutition sounds" audible over the posterior aspects of the left ninth and tenth dorsal vertebrae. The first is heard immediately after the act is produced by the downward projection of fluids. The second is produced by the passage of food through the cardia and is audible six or seven seconds after the act of deglutition. The presence of an obstruction is indicated by a modification in these sounds and by delay in their production (Meltzer[3]).

In *bougie examination of the esophagus three normal areas of narrowing* must be taken into account. The upper one is located at the beginning of the esophagus, the lower one at the hiatus, and the middle one corresponds to the bifurcation of the trachea. These are the points where foreign bodies are most likely to be arrested and where the greatest number of lesions are located.

Bougie examinations must be executed with gentleness. The English and French fabric and shellac bougies are best adapted for the purpose. When the presence of a diverticulum is suspected, a bougie fashioned like a Mercier's urethral catheter may be used to advantage. The angulated tip is expected to pass the entrance into the diverticulum. Before introducing the bougie, false teeth must be removed, and the presence of an aortic aneurism must be excluded. Flexible bougies are best inserted with the patient seated; the head should be slightly flexed, thus increasing the diameter of the entrance of the esophagus. The left forefinger holds the base of the tongue forward and the right hand carries the bougie against the posterior pharyngeal wall. The patient is instructed to breathe deeply and then the bougie is slowly advanced into the esophagus. The manuever is not a difficult one. The bougie should be sterile.

Congenital Malformations of the Esophagus.— Complete absence of the esophagus is very rare. *Total atresia* is also uncommon. Of the malformations, *separation of the esophagus* into two parts is the most common. The upper portion ends in a blind pouch which is distended, not unlike a diverticulum, and ends in the region of the jugulum. Occasionally it communicates with the trachea by means of a fistula. The lower rises from the stomach, is often very short and narrow and terminates in a blind end or opens into the trachea, the posterior wall of which may be absent. The malformation is due to developmental anomolies of the esophagus and trachea (Kreuter[4]). Children afflicted in this way become cyanotic when food is taken. The food flows out of the mouth and a fatal outcome ensues in about thirteen days. *Gastrostomy* and exclusion of the cardia have been practiced by v. Hacker.[5] The latter is intended to prevent food from gaining access to the trachea from below. If the child survives the operation, an attempt may be made to make an antethoracic esophagoplasty. If the blind ends of the incomplete esophagus lie sufficiently close together, an effort may be made to tunnel the space (Thomas[6]).

Injuries of Esophagus.— Injuries of the esophagus from within are produced by swallowing or introducing foreign bodies, which include bougies, coin catchers, and the esophagoscope. Injury from the esophagoscope occurs especially in cases of carcinoma, ulceration, and stricture of the gullet. Perforation of the esophagus is followed by invasion of the mediastinal or plural cavities by infectious processes.

Burns of the esophagus are discussed elsewhere (p. 1687).

Stab, incised and gunshot wounds involve the esophagus from without. They are not frequent and are usually a part of severe injuries of the contiguous tissues. In the neck a distinction between injuries of the pharynx and of the esophagus has no significance.

In the neck the trachea is usually injured at the same time. In not a few instances the thyroid gland and the large vessels and nerves are injured, especially in gunshot wounds. In the latter instance, the spine and the spinal cord are often involved. The esophagus alone is liable to be injured in stab and in gunshot wounds.

In *gunshot wounds* of the neck, the esophagus may not be opened, but may be perforated from ulceration of a severely contused area. A gunshot wound of the esophagus is always a severe injury. This is ascribed, in part, to the coincident complications, of which infection of the mediastinum is not the least common. This would seem to justify exploration in doubtful cases.

Incised wounds of the esophagus occur most often in connection with attempted suicide. Unless the wound is very small the egress of food makes the diagnosis. Simultaneous injury of the trachea has already been taken up (p. 1705).

The *treatment* of choice consists in immediate closure of the esophageal wound by suture. For the purpose, a double row of Lembert sutures is used. This is imperative, irrespective of whether the outer wound is closed or not. Even though complete primary union is not obtained, the period of healing is much shortened. When the trachea and esophagus are completely divided, circular repair by suture is indicated.

External esophageal fistulae follow isolated injury of the esophagus and the simultaneous injury of the food and air passages in the neck. External esophageal fistula is rare. On the other hand, a communication between the gullet and trachea is not uncommon. The former usually heals spontaneously. Simultaneous injury of the esophagus and trachea rarely leads to the formation of stricture, but fistula formation is very likely to follow. Of these, *laryngopharyngeal fistula,* with one or two external openings, is the most common. In these cases permanent use of the tracheotomy tube may be necessary. These external fistulae must not be confused with the persistence of an internal communication between the esophagus and trachea following injury.

The *treatment* of *external esophageal fistula* becomes necessary when the opening persists; this is the case when the mucosa is adherent to the opening in the skin. Closure may be attained in cases of moderate degree by refreshening the edges of the opening and subsequent suture When there has been much loss of substance, plastic repair of the defect may be necessary. In the latter instance, temporary gastrostomy possesses decided advantages. *In cases of simultaneous fistulae of the gullet and air passage* the technical difficulties pertain to the latter. In any event, an attempt should be made to close the opening into the esophagus first. In *esophagotracheal fistulae* the lesion must be widely exposed and repaired separately (v. Hacker[7]).

Injuries of the thoracic esophagus do not present a distinct clinical picture unless it happens that food emerges from the external wound. As a rule, the injury occurs in connection with an injury of other parts or organs, especially of the lungs. In the latter event, the tissues are emphysematous. The *diagnosis* is obscure, although the vomiting of blood, localized pain during deglutition, and perhaps the esophagoscopic picture may lead to the correct conclusion (Hochenegg[8]). The

treatment is not satisfactory. A temporary gastrostomy is justified on the ground that the undisturbed esophageal lesion may heal. Tamponade (with a string attached) of the esophagus through the esophagoscope has been suggested by v. Hacker.[5] Complicating mediastinal and pleural infections must be treated by aspiration and drainage (Part XV, chap. v).

Rupture of the esophagus occasionally occurs spontaneously. However, an underlying pathological condition may exist without being discovered (Mackenzie[9]). A certain number of cases are undoubtedly of traumatic origin, e. g., those resulting from violent vomiting.

Perforation of the esophagus, aside from invasion of the tube from without, occurs in connection with ulcerative and other destructive processes, such as tuberculosis, carcinoma, etc.

Esophageal hemorrhage is likely to appear in connection with almost all of the pathological processes occurring in this region and is of importance chiefly because of the necessity of differentiating it, when feasible, from gastric bleeding. Persistence of the hemorrhage demands temporary gastrostomy. Oral or retrograde tamponade may be made.

Foreign Bodies in the Esophagus.— Jackson's[1] résumé of the character of foreign bodies that have lodged in the esophagus leaves little to the imagination. From a practical standpoint this includes anything that may be forced past the entrance of the esophagus by the act of deglutition or by manipulation. As an illustration of the futility of any effort to go into detail in this connection, one may mention the case of an insane person who swallowed a copper crucifix (quoted by v. Hacker[5]). The religion of the patient is not stated. In a general way, foreign bodies may be divided into those with rough, pointed or sharp surfaces which cause injury, and those that are smooth and cause only obstruction.

The *points at which foreign bodies are arrested* depends upon the size of the latter. Small pointed objects may be fixed at any point of the gullet; very large ones rarely pass the isthmus, but remain in the pharynx. However, the former may be caught in the mucosa of the latter. Bodies of moderate size that pass the pharynx are most likely to be arrested at the *three points of normal narrowing.* By far the largest number of bodies are arrested at the thoracic aperture; those located below are usually forced downward by swallowing firm articles of diet or by the bougie. Those passing farther down are

arrested at the hiatus, almost never at the cardiac opening into the stomach. When the hiatus is passed the body enters the stomach.

The *symptoms of foreign bodies in the esophagus* depend upon their location, the distortion they cause in the gullet and the degree of consequent pressure upon neighboring organs (larynx and trachea) and upon whether they provoke ulceration and perforation or not. A body located behind the larynx causes respiratory disturbances.

In complete high obstruction, food is immediately regurgitated; in low obstruction, it is retained for a brief period of time. Sharp pain upon deglutition indicates that the foreign body has impinged upon the wall of the gullet. As a rule, patients are able to swallow fluids.

The *consequences* of the lodgment of a foreign body in the esophagus also differ widely. As a rule, they are serious unless relief is afforded by extraction, by offending agent's entering the stomach, or by its being expelled by vomiting. Blood is expelled through the mouth only when the body is located high up; the bleeding is rarely severe. Abscess formation is not uncommon. Ulceration, perforation, and infection of contiguous parts form a frequent sequence of events. Secondary invasion of this sort of the air passages leads to a menacing condition of affairs. Involvement of the mediastinal spaces, the pleura and the lung occurs in a certain proportion of the cases (Gerster[10]).

The *diagnosis* may usually be made on the symptoms. In their absence, the statements of the patient may not be relied upon. A more or less complete physical examination with negative findings has, on more than one occasion, terminated in discovering supposedly swallowed false teeth on the floor of the patient's bathroom or elsewhere. The pharynx and its vicinity may be examined directly. Large bodies located in the cervical region may often be palpated through the tissues of the neck. Secondary inflammation gives rise to pain and tenderness in this region. Rapid increase in the size of the thyroid gland (acute strumitis) often attends the presence of a foreign body in the cervical portion of the esophagus. It is ascribed to absorption of infectious substances.

The *Röntgenographic examination* is of the greatest value. Care must be taken in interpreting the negatives, so that calcareous deposits are not mistaken for foreign bodies. If the radiographic findings are positive, the passage of the bougie is unnecessary or perhaps unjustified, as the body may change its position at any time. Efforts at extraction must be made immediately after the radiographic examination.

Extraction of smooth, moderate sized bodies (coins) has been accomplished with the umbrella sound under the guidance of the fluoroscope (Heurad[11] — twenty-one cases). Careful sounding of the esophagus is of diagnostic value, although its field of usefulness has been much restricted by the radiographic and esophagoscopic examinations. The first should be used only when the last two are not available. A flexible (non-metallic) bougie should be employed for the purpose.

Removal of Foreign Bodies.— The removal of foreign bodies from the esophagus may be divided into *bloodless* and *bloody* methods. The former is accomplished by means of the *esophagoscope,* and, if

Fig. 844.— Röntgenogram of Foreign Body (safety pin) in Cervical Esophagus (Harlem Hospital case).

this is not available, by *direct instrumentation.* By both methods, the body may either be withdrawn or forced into the stomach.

Extraction by means of the esophagoscope is the most desirable method and often succeeds after other means have failed. It is especially indicated when the body is located above a stricture, in which event instrumentation is not successful in moving it in either direction. Killian[12] has succeeded in reducing the size of a prosthesis with the galvanocautery, after which it was readily pushed into the stomach. This has also been accomplished with the crushing forceps.

Direct instrumental removal through the mouth without esophagoscopy is likely to be attended with considerable danger. The danger is somewhat less when the body is smooth and flat. The purpose may

be accomplished by means of forceps or by instruments intended to pass the object and remove it as they are withdrawn. Of these instruments, the expanding probang (Fig. 845) is universally used. The instrument is introduced in its conical form. After passing the object the portion armed with twisted bristles is caused to expand and upon its withdrawal is expected to bring the offending substance with it. Small objects may often be removed in this way. Nevertheless, the method is very properly regarded as "a gamble." *Forcing bodies into the stomach* by means of indirect instrumentation is permissible when the obstruction is caused by soft substances, such as meat or potatoes, or by smooth, hard substances. An ordinary flexible bougie may be used for the purpose.

Operative methods are indicated when the others have failed or are primarily contraindicated. They may be divided into *pharyngotomy, trachelotomy lateralis, gastrotomy,* and *thoracic esophagotomy.*

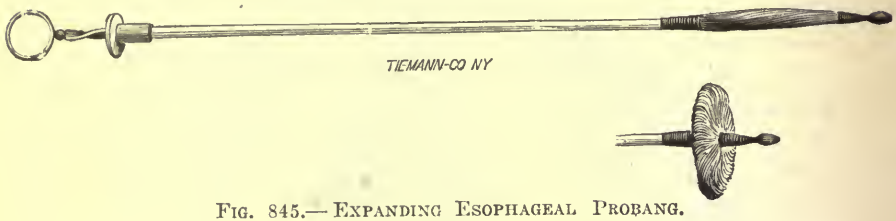

FIG. 845.— EXPANDING ESOPHAGEAL PROBANG.

Pharyngotomy is described in connection with the surgery of the larynx (p. 1695).

Lateral trachelotomy for exposure of the esophagus, i. e., without opening the latter, is practiced when it is possible to manipulate the foreign body in the pharynx and extract it — the advisability of which measure goes without saying. If this is not feasible, the esophagus is opened. The *incision* is made on the *left side* and corresponds in every particular to that employed for exposure of the common carotid artery, except that the omohyoid muscle is usually divided. The sheath of the vessels is drawn outward and the thyroid gland is drawn toward the median line. If necessary, the inferior thyroid artery, as it passes behind the carotid, is divided between two ligatures. The esophagus is readily identified by its longitudinal fibers, or the bulging foreign body very often discloses its location. The gullet is now opened, fixed with suture loops, and the foreign body extracted with the forceps. The esophageal wound is sutured only when its edges are not traumatized by the manipulations. The repair is made with

a double row of Lembert sutures. Complete primary union is rare. In either event, the external wound is drained. The establishment of a temporary esophageal fistula is necessary only in the presence of infection. A tube is immediately introduced into the stomach through the nose for the purpose of nourishing the patient. Sterile food is given for eight days. Permanent esophageal fistula is exceedingly rare.

Gastrotomy for the purpose of removing a foreign body from the esophagus is employed when the body is located 25 to 27 cm. below the teeth. The use of the esophagoscope has made this a rare operation. The *abdomen* is opened through the left rectus muscle; a purse string suture is introduced into the anterior wall of the stomach as near the cardia as possible; within this a small incision is made; the finger is introduced into the esophagus and the foreign body is "fished out." If this does not succeed, a string is passed into the stomach from above by means of a slender bougie. The former is delivered through the abdominal wall and a pledget of gauze tied to its oral end which is drawn downward; the maneuver is expected to drag the body into the stomach. The rule of the day is to expose the stomach freely, open widely in a longitudinal direction, introduce the gloved hand, and achieve the extraction with the fingers (Wood[13]). *Gastrotomy* is employed for the removal of foreign bodies located in the grasp of a stricture when the latter is to be simultaneously divided by the string method.

In thoracic esophagotomy dorsalis the gullet is approached through the posterior mediastinum. The method is very complicated and has, as yet, not proved satisfactory. The newer methods of opening the thorax under differences of pressure hold out a certain degree of hope for the future in this connection. See Surgery of the Thorax, Part XV, chap. vi.

The *treatment of the complications of foreign bodies in the esophagus* is of importance. *Bleeding* from the traumatized gullet or the result of ulceration demands immediate external esophagotomy and tamponade of the bleeding zone after the body has been removed. The *prognosis* is unfavorable, especially if the bleeding occurs from ulcerative perforation of a large vessel in the neck (Gerster[10]). The treatment of *retropharyngeal abscess* does not differ from that employed when the process is due to other causes. This is equally true with regard to a complicating *phlegmon of the neck*. It is of practical value to note that *purulent pleuritis, pneumonitis,* etc., may begin

in the connective tissue surrounding the esophagus and that the process may extend to the neck, thus forming part of an abscess cavity of enormous dimensions. The advent of these processes is attended with the clinical picture of a septic process. Its localization must *per force* be established by painstaking examination of the patient. Evacuation of the pus is of course imperatively indicated and is difficult only when the mediastinum is invaded.

Inflammatory Diseases of the Esophagus.— Acute and chronic catarrhal inflammatory diseases of the esophagus are of surgical importance only in as much as the pathological processes with which they are attended must be taken into account in interpreting the esophagoscopical picture. Chronic inflammations often complicate esophageal diverticula and esophageal spasm. The importance of *necrotic* and *diphtheritic* inflammations lies in their tendency to be followed by the formation of strictures. *Phlegmonous esophagitis* begins in the submucosa and occasionally may be *diagnosticated* when dysphagia is attended with the symptoms of fever which suddenly clear up after pus is vomited. Suspicion of the condition would seem to justify the careful introduction of the esophagoscope, through which the area might be incised.

Toxic or corrosive esophagitis, which follows the ingestion of corrosive substances, is of surgical importance because the swelling of the tissues may demand gastrostomy and later because of the sequential strictures that are formed. In severe cases in which the stomach is involved, the question of jejunostomy may come up.

Ulcers of the esophagus are due to various causes, of which tuberculosis and lues are the most frequent.

Luetic ulcers are comparatively rare; they occur most often in the upper portion of the esophagus. The process is, usually, primarily gummatous. This breaks down, and the repair is followed by more or less narrowing of the lumen of the gullet. Their presence is discovered *with the esophagoscope* and must be differentiated from tuberculosis, actinomycosis and carcinoma. The therapeutic test is valuable in this connection.

Tuberculous ulcers have only recently been recognized. They occur in connection with tuberculosis elsewhere in the body, especially that of the air passages (inoculation from swallowed sputum). The bibliography is collected by Gardère[14] and Guisez.[15] The former has collected 70 cases.

Actinomycosis of the esophagus should be borne in mind in connection with ulcerative processes in this situation (Garde[16]).

Peptic, or round, ulcer of the esophagus develops, as is to be expected, at its lower end. It is of importance because of the possibility of the development of pleuritis, peritonitis, etc.

Refinements in diagnosis in connection with the character of ulcerative processes in the esophagus have been made possible by the employment of the esophagoscope. There is no doubt that a closer scrutiny of cases of disturbances in deglutition will lead to a justification of this method of examination. The practitioner is asked not to dismiss his cases of so-called "heartburn" without taking the possibility of an esophageal lesion into account. Of course, the indiscriminate use of the method is to be deprecated. However, in properly selected cases it often clears up an obscure condition and makes relief possible.

Strictures of the Esophagus.— Of the causes which narrow the esophageal passage, those originating in connection with lesions of the walls of this organ are of surgical importance. These are genuine strictures and may be divided into *congenital, inflammatory, spastic, cicatricial and neoplastic.* However, in the differential diagnosis the surgeon must take into account the so-called *compression stenoses* which are due to enlargement or displacement of neighboring organs or parts. Of the various strictures of the esophagus, those caused by *carcinoma* and *corrosive agents* are the most frequent.

A conception of the conditions presented by strictures of the esophagus may be based upon those occurring as a sequel to the action of corrosive agents. These vary widely in extent, carrying out an accord with the degree of destruction of tissue, and thus correspond in a great measure to the strictures following the healing of other destructive processes. It should not require a great effort to visualize the processes concerned in the production of a cicatricial stricture of the esophagus from the viewpoint of the formation of scar tissue in any portion of the body — the urethra, the rectum, the skin, etc., especially when the deformations occurring in connection with extensive destruction of the last is borne in mind. That these deformations vary in extent and outline is to be expected, so that it is quite rational to classify them as *linear, semilunar, annular* and *tubular.* The annular and semilunar form, which offer great resistance to the passage of food, cause the upper segment of the gullet to dilate to a greater or less extent and its muscularis to become hypertrophied. The GREATEST DEGREE OF STENOSIS OF THE ESOPHAGUS OCCURS AT ITS BEGINNING, AT A POINT OPPO-

SITE THE BIFURCATION OF THE TRACHEA, AND IN THE REGION OF THE HIATUS. It seems hardly necessary to add that a stricture may cause *complete or incomplete stenosis of the esophagus.*

It is of practical importance to note that the dilated portion of the esophagus, in accord with the degree of stenosis, is the seat of chronic irritation, and the formation of diverticula is the outcome of retained articles of diet which undergo decomposition.

The main *symptom* of stricture of the esophagus consists in difficulty in deglutition which, in a general way, corresponds to the degree of obstruction. In slowly developing obstruction, the inability to swallow becomes gradually manifest; immediate regurgitation of ingesta argues for a high obstruction; their retention for several seconds indicates stenosis lower down. When the obstruction develops gradually, the patient is able to propel food into the stomach in small quantities at a time. The retention of a considerable quantity of food and its sudden expulsion, together with mucus, saliva, etc., indicates that the esophagus is dilated above the stricture. As a rule, fluids may be ingested after solids and semisolids are rejected.

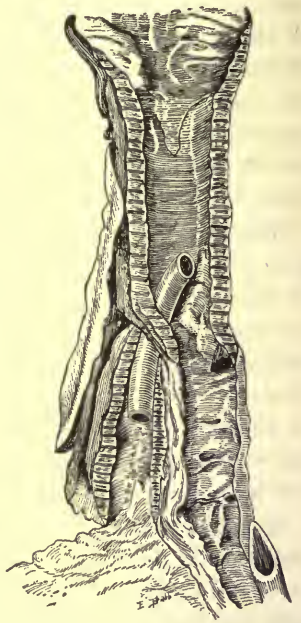

FIG. 846.—TUBULAR STRICTURE OF ESOPHAGUS EXTENDING FROM CARDIA TO BIFURCATION.

Tube in false passage in thoracic portion (v. Hacker).

The *diagnosis* of the presence and seat of a stricture is best established with the *bougie.* A description of the method has been given (p. 1781). After this is done, the esophagoscope may be used, with the view of determining the exact character of the obstruction. The Röntgenographic examination is very helpful.

The *differential diagnosis* between a cicatricial and carcinomatous stricture is readily made with the esophagoscope, especially when a section is removed and subjected to microscopical examination. The rarer forms of stricture already mentioned must be borne in mind, especially the luetic.

The *prognosis* in *cicatricial strictures* depends upon the degree of stenosis and upon the extent of the surface involved. Restitution to

the normal caliber of the esophagus is never attained; a functionally adequate lumen is as much as may be expected, and this only when dilatation is indefinitely persisted in. If the latter is omitted, the stricture recontracts.

The *treatment* of esophageal strictures (aside from those located in the cervical region, which may occasionally be resected, and those near the cardia, which are treated by interthoracic esophagogastrostomy) aims at dilatation of the stenosis. The two methods employed are *bloodless dilatation* with bougies and the *operative treatment*.

Fig. 847.— Esophageal Bougie Introduced over Filiform Guides (v. Hacker).

Dilatation is ushered in by the examination with the bougie, which determines the degree of narrowing and gives an index of the extent of the necessary stretching. The method is very simple and consists in the successive introduction of increasingly larger bougies. Patience, persistence, and gentleness on part of the surgeon, and coöperation on part of the patient are essential to success. At first, the bougie is left *in situ* 5 to 10 minutes; later, for half an hour. Dilatation need not be carried beyond 13 to 14 mm. in diameter. In narrow stricture a filiform guide may first be inserted and a tapered hollow bougie introduced over it (Fig. 847). A description of the refinements of the instrumentarium cannot be included here. While the esophagoscope gives valuable information with regard to the character and extent of the process under consideration, it does not add anything to the technic of the bougie treatment of strictures. As a rule, the obstruction lies at a point beyond the area of the lesion made visible by this instrument. The local and general use of *fibrolysin* has not yet proved of value.

The Operative Treatment of Esophageal Strictures.— Caustics may be applied to strictures through the esophagoscope. The appli-

cation of the corrosive fluid or the electrocautery is justified only in ulcerative stenoses or when a stricture is valvular in shape. The method must be immediately followed by bougie dilatation.

Electrolysis is employed by introducing through the stricture a metal sound which corresponds to the negative pole of the current; the positive pole (a metal plate) is applied to the anterior thoracic wall and an electric current of about 15 ma. is allowed to flow for from 50 to 60 seconds. The method seems to possess some value (Newmann;[17] see also report of Guisez[15]).

Internal esophagotomy by means of cutting instruments (on the order of the urethrotome) introduced from above is no longer practiced because of the dangers coincident with the procedure. Discission of a diaphragm-like stricture through the esophagoscope is permissible. Guisez,[15] who had two fatalities in seventeen cases, has abandoned the method in favor of electrolysis.

External cervical esophagotomy, i. e., opening the gullet in the neck, is practiced when the lesion is situated at this portion of the tube or is resorted to in order to make those lower down more readily accessible. The exposure is made for the purpose of (1) *extirpation of a stricture;* (2) *discission of a stricture;* (3) *establishment of an esophageal feeding fistula.* When the stricture is located below the thoracic aperture (more than 20 cm. below the teeth in the adult), cervical esophagotomy or temporary esophagostomy is practiced for the purpose of *dilating the stricture* or as a step in *combined esophagotomy.* The *technic* of esophagotomy is similar to that described in connection with removal of a foreign body. After the esophagus is opened, the stricture is divided in a longitudinal direction. The external wound is tamponed so that a *temporary esophageal fistula* is established. The lips of the wound in the gullet may be sutured to the skin and a tube passed into the stomach. The fistula may be used as a permanent means of feeding the patient in cases of impermeable stricture or in carcinoma.

In *combined esophagotomy* the gullet is opened in the neck above the stricture and the latter is divided with a herniotomy knife passed through the opening. A tube is introduced and left *in situ* for several days. The method is used when the stricture is located below the level of the tracheal bifurcation. It has not found favor.

Complete circular resection of a strictured segment of the cervical esophagus is indicated when the exposure is adequate and the stenosis is impermeable. The ends of the divided gullet are united by circular

suture. The operation is an extensive one. In order to prevent infection of the neck wound, temporary gastrostomy is advisable.

Gastrotomy for retrograde dilatation of esophageal stricture is rarely practiced at this time.

Gastrostomy is frequently employed in connection with obstinate strictures located deep in the esophagus. However, it is indicated only in cases in which the stricture remains impermeable to efforts directed from above (v. Haeker[5]). The method is used for retrograde dilatation of the stenosis, especially when inanition is threatening, as the fistula permits of feeding the patient. The fistula should be made as small as possible and should be arranged so that spontaneous healing occurs after the stricture is sufficiently dilated. After the gastrostomy opening has persisted for several weeks, it is often possible to pass a filiform guide into the stomach from above and withdraw it from below, or the reverse. Once this is accomplished, the use of the hollow bougie is comparatively easy. Various ingenious methods of establishing communication between the mouth and the fistula have been devised; of these the string method (Abbe[18]) is as satisfactory as any. The string, with a small ball attached, is swallowed and "fished out" of the stomach. The introduction of a bougie through the gastric fistula into the esophagus and retrograde dilatation are facilitated by the use of the gastroscope, which is not unlike the cysto-urethroscope in construction. In a certain number of cases esophageal instrumentation is made more effectual through a cervical fistula established for the purpose. IN ALL CASES IN WHICH ORAL DILATATION DOES NOT SUCCEED, AN EFFORT MUST BE MADE TO ACCOMPLISH THE PURPOSE THROUGH A GASTROSTOMY (not a gastrotomy) OPENING.

Transpleural esophagogastrostomy is an exceedingly complicated operation designed for the resection of portions of the thoracic esophagus. The thorax is opened in the manner described in Part XV, chap. vi (Meyer[19]).

Transperitoneal esophagogastrostomy is another difficult operation devised for the purpose of excising strictures located near the hiatus. Of the various methods, which include resection of ribs and the formation of thoracic osteoplastic flaps, that of transdiaphragmatic approach would seem to hold out the best chances of success (Heyrovsky[20]). The surgeon should be certain that the indications for an operation of this magnitude are clearly defined before exposing the patient to the attendant risks.

EXCLUSION OF THE ENTIRE THORACIC ESOPHAGUS — ANTETHORACIC

Esophagoplasty.— In cases of impermeable stricture an effort has been made to exclude the thoracic esophagus by antethoracic esophagoplasty (v. Hacker,[21] Meyer,[22] Trendelenburg[23]). The methods of accomplishing the purpose are:

1. Antethoracic dermato-esophagoplasty.
2. Antethoracic entero-esophagoplasty.
3. Antethoracic dermato-entero-esophagoplasty.

In *antethoracic dermato-esophagoplasty* (Bircher[24]) the cervical esophagus is delivered through the neck and connected by suture with a tubular canal fashioned from two layers of the skin of the anterior thoracic wall. This *dermatogenous* tube is connected with the anterior wall of the stomach by drawing a fold of the latter through a wound in the anterior abdominal wall and making an end to side anastomosis. The likelihood of failure is, to say the least, not remote.

Antethoracic entero-esophagoplasty, esophagojejuno-gastrostomy (Roux[25]), consists in isolating a loop of jejunum, implanting its anal end into the stomach and later connecting it with the cervical esophagus. See Bibliography of v. Hacker.[5]

In *antethoracic dermato-entero-esophagoplasty* (Wullstein[26]) the upper segment of the vicarious tube is made, as in the first method, and this is connected with a loop of jejunum, which in turn is anastomosed to the jejunum, excluding the thoracic esophagus and the stomach from digestive function (v. Hacker[5]).

Dilatation of the Esophagus.—Dilatations of the esophagus, as already stated, occur above the site of stenosis of various kinds. They also develop when there is no discernible lesion. While no doubt most of the cases are *congenital* in origin, *disturbances of innervation* would also seem to be a causative factor (*cardiospasm*). The spasm of the cardia may be reflex. The vagus, so often accused, would seem to be exonerated by the investigations of Gottstein[27] and of others.

The *symptoms* are those of esophageal obstruction plus "cramp in the gullet." These disappear as soon as the esophagus is emptied downward or by regurgitation of its contents. An attack may last for several hours. The food retained undergoes decomposition, which provokes foul smelling eructations. Many cases go to autopsy without being recognized. On the other hand, cardiospasm and dilatation of the gullet have been diagnosticated in cases of carcinoma and ulceration in this situation. The obstinate persistence of so-called gastric indigestion should arouse suspicion of the condition.

Examination with the bougie is indicative when the instrument may

be carried over wider than normal excursions in a rotary direction. Very often a large bougie enters the stomach, while a smaller one is arrested in a smaller sacculated dilatation. As a rule, unaltered food may be aspirated. The quantity of returned fluid is indicative of the degree of dilatation. It may be as much as a liter. *Esophagoscopic* and *Röntgenographic* findings are of determining diagnostic import.

In the *treatment* the first consideration is to prevent the accumulation of food. This is accomplished by lavage. Progressive dilatation of the cardia is at times curative. A *temporary gastrostomy* makes the bougie treatment more effective. A Symonds' bougie may be left *in situ* over night. The application of electrolytic stimulation to the spasmodic cardia and the administration of atropin are advised by Kaufmann and Kienböck.[28] If the cardia is accessible to the bougie, its dilatation may be practiced (Lerche[29]).

Posterior mediastinotomy and plication of the esophagus has been performed by Meyer[22] in several instances. It is a procedure of magnitude. *Forcible dilatation of the cardia* through a gastrotomy opening has proved effectual (Erdmann[30]). *Subdiaphragmatic esophagogastrostomy* is practiced by Heyrovsky.[20]

Diverticula of the Esophagus.— The term *diverticulum* is applied to a restricted sacculation of the esophagus. When the protrusion is lined by mucous membrane, the term *true diverticulum* is used; when the former is absent the term *false diverticulum* is employed. True sacculations are divided into *pressure and traction diverticula.*

Pressure diverticula are subdivided, according to their seat, into those located at the pharyngo-esophageal junction and those more deeply situated.

Pharyngo-esophageal diverticula are located, as their name implies, in the neck, and originate from the posterior lateral (left) wall of the gullet at a level with the cricoid cartilage. They appear as pear shaped sacs and may reach a length of 13 cm., thus obtruding into the thoracic cavity. The opening communicating with the esophagus varies in size. The diverticulum usually possesses a thickened wall, which consists of the hypertrophied layers of the esophagus.

The *causation* is undoubtedly pressure from within. However, there is no doubt that the congenital lack of muscle fibers at the posterior aspect of the pharyngo-esophageal junction is the responsible underlying causative factor. Any condition of the neck, such as struma, which favors the retention of food in this situation, may be regarded as a determining cause of the condition. According to Lotheisen,[31]

nervous persons subject to spasm of the pharynx are likely to develop the lesion.

The *symptoms* do not appear until the sacculations reach a certain size. These may consist simply in frequent expectoration of tenacious mucus, which often leads to the belief that a lesion of the larynx is present. However, most cases are afflicted with *eructations, nausea, and vomiting* of moderate quantities of ingesta. The last symptom is usually regarded as evidence of gastric disturbances. As the

FIG. 848.—RÖNTGENOGRAM OF PHARYNGO-ESOPHAGEAL DIVERTICULUM.

diverticulum increases, all the ingesta are regurgitated (Butlin[32]). At this time swelling becomes palpable at the left side of the neck. This may often be emptied by pressure, the food being forced back into the mouth. When the diverticulum cannot be emptied, the food decomposes and gives rise to distress and vomiting. At times the swelling in the neck gives the impression of a goiter.

The *diagnosis* may often be made by the history of gradually increasing difficulty in swallowing, and regurgitation of unaltered food. This is, of course, strongly supported by intermittent swelling of the

neck. Examination with the bougie, especially when its end is palpable in the neck, is helpful (Butlin[32]). The diagnosis may be made certain with the *esophagoscope*. Lotheisen[31] uses an especially constructed instrument for the purpose. The Röntgenogram is of signal service. (Fig. 848).

The treatment consists of *operative extirpation of the diverticulum.* When the patient is much reduced, this may be preceded by temporary gastrostomy. The technic of exposure is similar to that of esophagotomy cervicalis lateralis (p. 1794). The diverticulum is amputated and the opening closed with two layers of Lembert sutures, using a cuff of the adventitious sac for the purpose; the inverted neck of the sac forms a valve which obviates leakage (Lexer[33]). In a certain number of cases diverticula become the seat of ulcerations which later develop malignant disease (Pitt,[43] Starck[35]).

In *deep seated diverticula,* the clinical picture (except for swelling in the neck) does not differ from that described in connection with those located in the neck. As a rule, the diagnosis is doubtful until the esophagoscopic and Röntgenographic findings establish the true condition of affairs. No doubt a certain number of cases, at first regarded as gastric, upon closer study would be recognized as diverticula of the esophagus. This is shown by a routine series of complete Röntgenographic examinations of the entire digestive tract made by Caldwell (personal communication), whose untimely death prevented publication of his work in this connection.

Lavage of the diverticulum and *bougie dilatation of the normal tract* improves the general condition of the patient. The establishment of a feeding gastrostomy is justified. The operative approach is similar to that described in connection with stricture (p. 1797). Ablation of the sac is accomplished as in cervical diverticulum (Spivek[36]).

Traction diverticula rarely produce symptoms. They occur in connection with chronic inflammatory processes involving contiguous parts, organs, and tissues. Of these, tuberculous lymphadenitis is the most frequent; the cicatricial contraction coincident with the lesion drags out a portion of the esophageal wall which later becomes sacculated from internal pressure. The literature on the subject is not extensive. That of v. Hacker[5] may be consulted to advantage.

Tumors of the Esophagus.— Cysts, papillomata, fibromata, lipomata and myxomata of the esophagus possess more pathological interest than clinical significance.

The term *polypus of the esophagus* embraces pedunculated tumors

of various kinds, such as fibromata, fibrolipomata and myxomata, which arise in the submucosa and protrude into the lumen of the gullet. They are usually found at the pharyngo-esophageal junction and near the bifurcation of the trachea.

The polypi are, as a rule, pear shaped and rarely give rise to symptoms, unless they grow to a large size. In this event, the lesion presents the clinical picture of gradually increasing esophageal obstruction. The exact diagnosis may be made with the esophagoscope. Spontaneous avulsion of the tumor (during vomiting) has occurred (Göbel[37]).

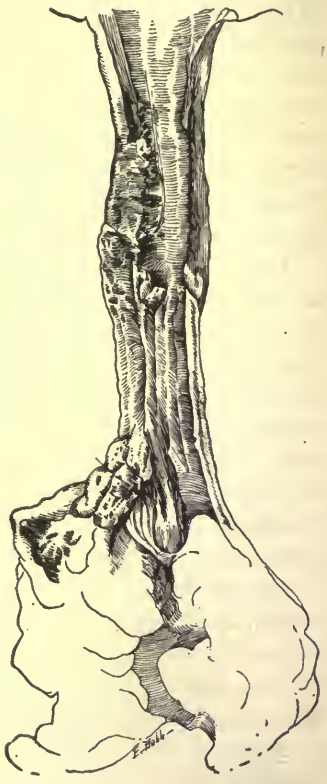

FIG. 849.— CARCINOMA OF ESOPHAGUS.

Carcinoma is by far the most frequent tumor met with in the esophagus. The growth may be *primary or secondary.* The latter occurs as the result of extension from neighboring tissues; actual metastases have not yet been reported.

Carcinoma of the esophagus is usually of the *flat cell* variety, although colloid and cylindrical cell also occur. They are usually situated at the three points already spoken of, i. e., opposite the cricoid cartilage, at the bifurcation of the trachea, and near the hiatus (Guisez,[15] Gottstein[27]). According to v. Hacker,[21] the level of the bifurcation is its favorite seat.

The *chief symptom* of carcinoma of the esophagus consists in difficulty in swallowing. As the growth infiltrates the gullet, it interferes with its contractility, so that deglutition is obstructed to an extent out of proportion to the degree of stenosis. The symptomatology of the affection is that of gradual obstruction of the esophagus, including *pain* and *spasm,* attended with gradual loss of weight out of proportion to the mechanical interference with deglutition. When the tumor is located in the neck, it may be palpated as a slowly increasing tender mass attended with enlargement of the lymph nodes.

The *diagnosis* takes into consideration the clinical picture as stated,

the age of the patient, and the esophagoscopic and Röntgenographic findings. The esophagoscope must be carefully introduced. If feasible, a section of the growth is removed for microscopical examination.

The *prognosis* is unfavorable, even after extirpation of the growth. This gloomy outlook has been somewhat illuminated in cases of carcinoma of the upper esophagus by the radical work of Gluck.[38]

Extirpation of carcinoma of the esophagus is practiced in the manner already described in connection with operations on the neck and resection of the esophagus for stricture (p. 1799). The mortality of transpleural extirpation is 100 per cent. The palliative operative procedures are taken up in connection with strictures of the esophagus. Of these, gastrostomy is the most satisfactory. The use of the bougie is a dangerous maneuver. *Local treatment* of the lesion through the esophagoscope and the use of radium are of doubtful value. See chapter on carcinoma, Part VI.

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PART XV

SURGERY OF THE THORAX

CHAPTER I

INJURIES AND DISEASES OF THE THORACIC WALL

The *congenital deformations* of the shoulder girdle and the vertebral column and their influence upon the contents of the thorax have been made the object of careful clinical study by the orthopedist (Chas. Fère et Schmid,[1] Ebstein[2]). This aspect of the problem is taken up in connection with the Surgery of Deformities, Part VIII.

INJURIES OF THE THORAX

Contusion of the Thorax.— Contusion of the thorax without apparent serious injury to the chest wall is of importance because of the injury to the thoracic contents likely to occur in this connection. These vary from a small rupture in a lobule of the lung to more or less severe trauma to the heart and great vessels. The *lung* and *pleura* are most often involved. The mechanism which produces the interthoracic injury is explained on the basis that the air does not escape through the glottis with sufficient rapidity to permit the thoracic contents to adapt itself to the forcible compression produced by the trauma (Koenig[3]).

The *clinical picture* varies with the degree of intrathoracic injury, being that of *hemothorax, pneumothorax* and *hemopneumothorax. Rupture of the diaphragm* allows the abdominal contents to prolapse into the thorax and this may be attended with the symptoms of *intestinal obstruction,* though not infrequently the condition remains undiscovered until autopsy. A certain number of cases develop so-called *contusion pneumonitis,* which runs a mild course unless an infection develops, and then the picture is that of *infectious pneumonitis* and all its attendant complications. *Rupture of the lung* may be attended with *traumatic emphysema* (p. 560). This may be limited to the face and neck and upper part of the chest and is explained by the fact that the veins in this region do not possess valves (Perthes[4]). *Hernia of*

the lung may occur without fracture of a rib. The mortality from severe contusions of the chest is high.

The *treatment* of contusion of the thorax varies in accord with the symptoms. Absolute rest in the horizontal position is essential. The administration of small doses of morphia is advisable. Shock is treated in the usual way (p. 945). Large effusions of blood are removed by aspiration. The value of *thoracotomy* for the purpose of arresting bleeding in subcutaneous rupture of the lung is not yet established. However, recent observations would seem to justify its employment in selected cases.

Concussion of the Thorax.— Trauma to the thorax *without any changes in its walls or contents* may be attended with a clinical picture to which the term concussion of the thorax is applied. As a rule, the condition is ascribed to *shock*. However, there seems to be no doubt that trauma applied to this situation is occasionally attended with results ascribable to *lowering of the blood pressure* and to direct irritation of the vagus. The causative force may produce direct concussion of the heart, which then presents the manifestations coincident with vagus irritation and is arrested in diastole. Persistence of lowered blood pressure indicates concussion of the depressor nerves and of the sympathetic nerve.

Menacing *symptoms* or a fatal issue following a blow upon the thorax, especially upon the precordial region, may, in certain instances, be properly ascribed, first, to heart failure, the outcome of intrathoracic irritation of the vagus, and, later, to the lessening of the tone of the peripheral circulation.

The *prognosis* is, as a rule, not unfavorable. The *treatment* consists in placing the patient in the recumbent position, followed by artificial respiration, and stimulation. Elevation of the limbs, with a view to increase the quantity of blood in the thorax (autotransfusion), is advised by Riedinger.[5]

Non-penetrating wounds of the thorax are of importance because of the necessity for differentiating them from the penetrating. In *stab wounds* and *gunshot wounds* this is not always easy. In doubtful cases drainage should be employed and the wound lightly tamponed. Embedded projectiles are best removed.

Of the *complications*, injury to the *internal mammary artery* is of great importance. The vessel runs about 1 cm. external to the edge of the sternum. As a rule, the pleura is punctured, but this is not by any means always the case. The wound must be enlarged and the bleeding

ends of the artery tied. If this is not feasible, tamponade for ten days may be practiced. The intercostal arteries are treated in a similar fashion.

Fractures of the sternum and ribs are taken up in Part IV, chap. iii.

INFLAMMATORY DISEASES OF THE THORACIC SKELETON

Hematogenous Osteitis and Periostitis of the Ribs and Sternum.—Acute osteomyelitis of the bones of the thorax follows the laws of localization laid down in the general part of this book (Part II, chap. ii). Excluding the vertebrae, the ribs are more often invaded by the bacteria of acute infectious diseases than are the other bones of the body. *Typhoid osteomyelitis* is very common.

Acute osteomyelitis of the ribs presents the *clinical picture* of an *acute infectious osteitis*. The local symptoms usually begin at the metaphyses. The *diagnosis* must take into account similar infectious processes of the soft parts. The *treatment* consists in early resection of the infected bone.

Typhoid osteitis and chondritis of the ribs deserve special mention. They usually develop during the convalescent period of so-called typhoid fever and may be a *mixed infection*. In a certain number of cases the typhoid bacillus is crowded into obscurity by the more aggressive pyogenic bacteria. While the process usually exhibits a tendency toward chronicity and presents a *clinical picture* not unlike that of bone tuberculosis, the domination of the mixed infection gives rise to acute exacerbations of the lesion.

Acute osteomyelitis of the sternum is of importance because of the danger of secondary involvement of the mediastinum. This urges prompt subperiosteal resection of the infected bone and the establishment of free drainage.

Tuberculosis of the ribs and the sternum may appear by itself or may be associated with tuberculosis of other bones of the body. The latter condition of affairs occurs in 70 per cent of the cases (Koenig[6]). A considerable number of cases are the result of extension from tuberculosis of the vertebrae, pleura, and lung.

The primary hematogenous focus usually appears as a localized process which soon undergoes caseation or sequestration. The superimposed soft parts are involved in the form of a cold abscess which breaks down, leaving one or more fistulous tracts communicating with the bone lesion.

Tuberculosis of the sternum presents a similar picture and very frequently extends to the sternoclavicular articulations. It shows a tendency toward sequestration, so that occasionally the entire manubrium breaks down. Perforation into the mediastinum occurs, but is rare.

The *clinical picture* of tuberculosis of the sternum and ribs is that of a chronic inflammatory process. In many instances a spindle shaped enlargement of the bone is the first evidence of the lesion. The lesion may remain at this stage for a long time. However, it usually breaks down, forms a cold abscess, and later establishes the usual sinuses.

The *diagnosis,* in the early stages of the disease, may not be clear, especially when the lesion is restricted to a small area of thickening of the affected bone. In these instances the microscope (aspiration), the Röntgenogram, serological examinations, and exploratory incision must be sequentially employed. When the lesion breaks down, the diagnosis is no longer in doubt. The *prognosis* depends upon the underlying causative factor.

The *conservative treatment* of tuberculosis of the thoracic skeleton *in children* is permissible. See Bone Tuberculosis, Part II, chap. xxiii. *In the adult,* radical excision of the focus is indicated.

Luetic inflammations of the thoracic skeleton appear with all the landmarks of osteochondritis luetica. However, there is little to differentiate the process from tuberculosis. Of the diagnostic methods, other than the serological and microscopical findings, the *therapeutic test* is the most valuable. The *treatment,* other than medication, consists in resection of the affected bone.

Actinomycosis of the thoracic wall presents the clinical picture of a hard, chronic, inflammatory, infiltrating process, usually attended with the formation of *multiple fistulae.* The *diagnosis* is made certain by the microscopical findings (Part II, chap. xix).

Intercostal Neuralgia.— The surgical significance of intercostal neuralgia, aside from that taken up in connection with the surgery of the spine (Part IX, chap. iii), lies in the causative relationship of pressure upon the nerves due to fractured ribs, osteomyelitis, tumors, etc., the removal of which affords relief. In a certain number of cases, in which it is not feasible to remove the cause, *extirpation of the nerve* is permissible. This is easily accomplished by lifting the nerve from its bed at the lower border of the rib and extirpating it in the manner described in connection with the superficial branches of the trigeminus. The uncertainty of the operative outcome has led to the employment

of the subcostal injection of 70 per cent alcohol or 1 per cent osmic acid solution — measures which are followed by relief in a large percentage of the cases.

Tumors of the Thoracic Wall.— Cysticercus cellulose is occasionally found in the musculature of the chest wall. It is usually not recognized until it breaks down.

Echinococcus appears in the soft parts and in the bones of the thoracic wall. It presents the picture of a slowly growing tumor, the nature of which must be determined by the microscopical examination of the aspirated contents. The presence of eosinophilia and an unexplained urticaria may lead to a suspicion as to the nature of the growth.

Nevi and epithelial cysts do not present any regional peculiarities.

Lipomata are the most frequent tumors of the chest wall, especially of its posterior surface. Solitary lipoma appears in the form of a pear shaped tumor, which is often pedunculated. It not infrequently undergoes degenerative processes, though malignant changes are exceedingly rare. Lipomata of the axillary region may contain rudimentary mammary gland tissue, in which event they become enlarged and tender during menstruation and pregnancy. Retromammary lipomata push the breast outward, sometimes to an astonishing extent. Tumors of this sort are easily extirpated.

Fibromata, pure neuromata, hemangiomata, and *lymphangiomata* do not present regional peculiarities. *Osteomata* and *chondromata* are very rare.

Chondromata usually arise at the costocartilaginous junction, and at the point of union of the manubrium with the gladiolus, or of the latter with the ensiform cartilage. They do not give rise to symptoms until pressure is made upon the intrathoracic structures. In the early stage of development they must not be confused with tuberculosis and lues. The differentiation is based on the Röntgenographic examination. They are easily extirpated.

Malignant Tumors of the Thoracic Wall.— Enchondroma can no longer be regarded as a benign tumor (Ribbert[7]). Enchondromata of the sternum and ribs are not recognizable as such, except upon microscopical examination. They appear in the guise of a rapidly growing tumor of the bone, which bears considerable resemblance to sarcoma.

Primary sarcomata of the thoracic wall begin in the *soft parts* and in the *bones*. Those of the *soft parts* are comparatively rare. They

are usually of the melanotic or small round cell variety, both of which soon form metastases and, even when extirpated early, have a tendency to recur. Most of the tumors spring from the fascia of the pectoralis major muscle. *Secondary sarcomata* of the soft parts occur in connection with sarcomatous processes located elsewhere in the body.

Primary sarcomata of the osseous structures of the thorax are of the usual periosteal and myelogenous, osseous and osteoid varieties.

Periosteal sarcomata appear as spindle shaped or nodular masses continuous with the surface of the bone; they are likely to be very malignant. Those of the myelogenous form are somewhat less so. The latter often give rise to persistent intercostal neuralgia, the cause of which is not apparent until the process breaks through the bone.

Sarcomatous ribs soon fracture. After this the process spreads very rapidly in all directions, invading the pleura, the lungs, the heart, etc.

Pressure *symptoms* do not appear until the process has invaded the thoracic cavity and are in proportion to the extent of involvement.

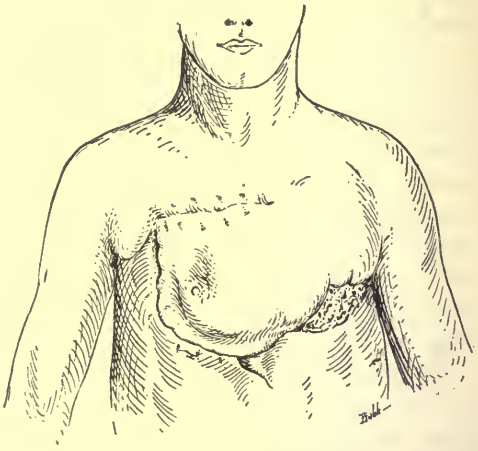

FIG. 850.— EXTENSIVE RESECTION OF THE THORACIC WALL.

Defect covered with flap from opposite side.

The *diagnosis* is not easy at first. It is based on persistent pain, increasing anemia, loss of weight, and the Röntgenographic findings. Ultimately the malignant character of the process becomes manifest.

Carcinoma of the thoracic wall, other than that developing in connection with carcinoma of the breast, is rare. Perhaps the so-called "*corset carcinoma*" may be mentioned in this connection. Occasionally carcinoma of the mediastinum extends to the skin and other soft parts of the chest.

The Technic of Resection of the Thoracic Wall.— The extirpation of malignant tumors of the chest wall without simultaneous removal of the contiguous pleura is rarely permissible. If possible, the probable extent of the operative measures should be determined before the

attempt at extirpation is made. This is true even in cases of tuberculous and luetic processes for relief of which the pleura and, perhaps, the lung must be invaded by the surgeon.

While it is undoubtedly true that the thoracic cavity has been, and is, constantly successfully invaded without the protection of differential pressure apparatus, it is also true that employment of the latter simplifies very much the technic of the procedure and markedly lowers the mortality. If the operation is executed without *pressure differences*, an effort should be made to suture the lung to the opening in the wall of the thorax, or a flap taken from the contiguous tissues may be turned or slid into the defect (Riedinger[5]).

When the principle of pressure differences (Sauerbruch[8]) is employed, the skin and musculature are divided down to the ribs (differential pressure 5 mm. Hg.); an intercostal space is rapidly opened and the entire sector of the thoracic wall, including the ribs and pleura, is quickly excised. The intercostal vessels are now tied and the necessary amount of lung tissue is extirpated. By increasing the pressure to 10 to 12 mm. Hg. the lung is forced into the defect in the chest wall and repaired by suture. The defect in the thoracic wall may be closed in the manner shown in Fig. 850. Thus the formation of a *secondary pneumothorax* is obviated. *Resection of the sternum* and invasion of the mediastinum is executed in a similar manner (p. 1854).

The acquired deformities of the thorax are taken up in Part VIII.

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CHAPTER II

PNEUMOTHORAX

As soon as contact between the surface of the lung and the thoracic wall is abolished, as happens when the latter is opened, the "retraction capacity" of the lung is brought into play and the organ is drawn toward its hilus (it is said to collapse) and a pneumothorax is established. If the opening in the thoracic wall persists, an *open pneumothorax* exists; when the external opening is spontaneously or artificially closed, a *closed pneumothorax* is present. A similar condition of affairs obtains when a solution of continuity in the lung occurs. The open pneumothorax is of especial surgical importance.

The degree of dyspnea attendant upon unilateral pneumothorax is in a measure dependent upon the elasticity of the mediastinal wall. If this is not sufficiently resistant (mediastinal flutter), the function of the uninjured lung is seriously interfered with (from pressure). Grave disturbances do not always appear as the result of the establishment of a pneumothorax. This has led to the erroneous belief that the condition is not dangerous. As a matter of fact, the reverse is true. Indeed, it is not improbable that the absence of serious disturbances is due to an anatomical peculiarity, such as adhesion of the lung to the parietal pleura or a particularly resisting mediastinal membrane (Sauerbruch[1]).

The Prevention of the Consequences of Pneumothorax.— When the thoracic wall is opened the disturbances consequent to pneumothorax may be abolished by grasping the lung with forceps and drawing it into the wound. This prevents fluttering of the mediastinal wall. When the thorax is accidentally opened during an operation, the wound is immediately sealed by suture. As already stated, after resection of the thoracic wall, the lung may be sutured to the edge of the defect. There is no doubt that a persistent, closed pneumothorax is especially susceptible to infection (Nötzel[2]).

A number of methods devised to obviate the persistence of pneumothorax found their circulation in the establishment of the principle of

maintaining the normal negative pressure, which prevented the lung from retracting (Sauerbruch[1]). The basic theory of the procedure is to keep the lung inflated from the bronchial tree by suction upon its surface. The desired end may be attained by lessening the pressure of air on the surface of the lung — *negative pressure* — or by increasing pressure on the bronchial tree — *positive pressure.*

It is certain that the methods prevent the development of pneumothorax. The lung on the opened side is made to functionate as a respiratory organ. In practice, the pressure is never sufficient to interfere with aëration of the blood. IN A GENERAL WAY, IT IS JUSTIFIABLE TO STATE THAT THE EMPLOYMENT OF DIFFERENCES IN PRESSURE PREVENTS THE CONSEQUENCES OF PNEUMOTHORAX IN A HARMLESS MANNER.

The *Sauerbruch cabinet* may be described as follows:

The cabinet is made of metal and glass and large enough to accommodate an operator and two or three assistants. The patient's body lies upon a table with the head protruding through an opening in one side, guarded by a rubber collar which fits closely to the neck. The abdomen and lower extremities are covered by a canvas covered rubber sac which communicates by means of a tube with the outside air. This maintains the ordinary atmospheric pressure upon all parts except the thorax and upper extremities. Narcosis is administered under atmospheric conditions. The cabinet is well lighted and is provided with telephone connection. The air is exhausted from within and negative pressure produced by an electric suction pump. Later, a small, airtight anteroom was added, communicating with the main room and outside by airtight doors. As the suction pump communicates with this room, additional instruments may be called for, placed in this room, and, later, brought to the main operating room without affecting the air pressure. See Keen's Surgery, v. iii. When sufficient air is withdrawn from the cabinet to render the negative pressure equal to 10 to 12 mm. Hg., both pleural cavities may be opened without collapse of the lungs. With the aid of this apparatus extensive mediastinal and esophageal operations may be performed, both pleural cavities being opened without any of the deleterious effects or sequelae of pneumothorax. Sauerbruch[3] reports sixteen intrathoracic and mediastinal operations with most encouraging results, the fatalities in no instance being attributable to pneumothorax. Sauerbruch[4] at this time regards the employment of positive pressure face masks and intratracheal apparatus as of the greatest practical value in general sur-

gical practice. He says that "the positive pressure apparatus accomplishes the purpose as well as the negative pressure cabinet."

F. T. Murphy[5] uses an airtight helmet, inclosing the patient's head and neck, in which ether is administered and air pressure increased and maintained by water displacement. Green[6] produces artificial respiration by a positive pressure plan, in which he uses the Hans Weger pump and his own etherizing apparatus and an intralaryngeal cannula. Janeway[7] has devised a very elaborate etherizing apparatus, by which artificial respiration is maintained with the aid of an intratracheal tube (Fig. 86). Matas,[8] Fell,[9] Meyer,[10] and Meltzer[11] overcome the effects of pneumothorax by a tightly fitting tracheal cannula or intubation tube and some form of air pump or bellows.

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CHAPTER III

PENETRATING WOUNDS OF THE THORAX

The severity of penetrating wounds of the thorax is proportionate to the visceral damage and the elements of infection. Those involving the great vessels, the mediastinum, the roots of the lungs, the heart or the pericardium are usually promptly fatal. In diaphragmatic involvement the abdominal contents are likely to be invaded. The introduction of pyogenic baceria at the time of injury results in the development of pyothorax. This may arise in connection with infection of the chest wall, or the infection may originate in the lung. Pneumonitis is a grave complication, regardless of the extent of the visceral injury.

Gunshot Wounds of the Thorax.— Gunshot wounds of the thorax usually involve the thoracic contents. Lilienthal and a number of coworkers make the following statement as expressing their conclusions on war surgery: "The thorax is no longer a region where only a few dare to travel. Resection of a part, of a lobe of lung, suturing of wounds of the lungs and ligation of vessels are now common procedures. Thoracic explorations are made in doubtful cases, as are exploratory celiotomies. At the same time, the conservative treatment of a certain variety of cases has been emphasized." In this connection Bastianelli[2] says "Truly, for small puncture wounds produced by rifle bullets without severe bone lesions, the principle of expectation may be adhered to, at least with regard to life, but not always, if function is to be considered. Expectant treatment should be followed more rarely in shrapnel wounds, and even less frequently for shell splinters." Experience in previous wars shows that the high velocity rifle bullet wounds offer a favorable prognosis in comparison with the older ones of low velocity and large caliber. In the latter, fragments of splintered ribs and clothing were introduced into the wound. This gave a much larger percentage of infections with greater laceration of lung tissue and severe hemorrhage and, because of the decreased velocity of the missile, it frequently remained embedded in visceral tissue. Because it forces ragged

pieces of metal into the body the shrapnel used during the late European war has caused severer wounds than the old slow velocity bullet. This is in a measure equalized by the present treatment of this type of wound.

The *symptoms* of gunshot wounds of the thorax depend upon the direction of the penetration. Severe *shock* may exist with but slight visceral injury, and *vice versa*. The signs and symptoms of *internal bleeding* are indicative of the involvement of large vessels. *Dyspnea* may be due to air hunger from this cause, but is also indicative of *pneumothorax* or *hemothorax*, pericardial or heart involvement.

The *diagnosis* is not difficult. In the absence of subjective symptoms, examination reveals pneumothorax, emphysema, etc. Localized pain during respiration, with cough, is indicative of pleural involvement. Marked dyspnea, increased respiration, with absence of normal respiratory sounds and hyperresonance, indicate the entrance of air into the pleural cavity. Hemorrhage generally causes flatness at the base of the lung with loss of fremitus and respiratory sounds. Emphysema at the root of the neck is indicative of lung injury. Bloody, frothy expectoration after attacks of coughing is diagnostic of a similar condition; inrush of air may be heard on auscultation. In locating foreign bodies, X-ray and fluoroscopic examination are indispensable.

The *prognosis* is not necessarily unfavorable, the outcome depending upon the site of the injury and the visceral involvement. Injury to the heart and great vessels is usually immediately fatal. Operative treatment has greatly lessened the mortality. Duval[3] says "Altogether our personal statistics from the battle of the Somme, where, excluding operations of urgency, cases were treated medically, show a mortality of 20 per cent in three hundred cases. In a later series, subjected to operative treatment, of 136 cases (18 urgent and 118 not), the general mortality was 9 per cent. It will be seen, therefore, that surgical treatment lowers the mortality very considerably, and that when septic complications do occur, they are less serious. In the above cases we had eleven cases of emphysema with one death."

The menace of lung injury (except in large lacerations where the immediate prognosis is unfavorable) lies in infection and subsequent development of empyema, abscess and gangrene. The possibility of infection is always greater from an external wound than from one of the lung. If the wound is sealed, pneumothorax adds no great danger to the problem, as the air is readily absorbed. On the other hand, blood is taken up with much less certainty.

Crushing injuries with compound fractures of the sternum and ribs are especially dangerous. Lilienthal[1] says "The complication of fractured ribs may be considered one of the most dangerous of war wounds of the thorax. It almost invariably leads to infection, especially when numerous spicula have penetrated the lung."

The *treatment* of gunshot wounds of the chest is divided into the *immediate* and *subsequent*. The former includes the routine care of shock and hemorrhage and the latter the prevention of infection. Morphin is a useful agent when used with discretion. The external wound should be cleansed and a dry sterile dressing applied. The superficial wound is sutured only when the soft parts are extensively lacerated, and then must be drained. Fractured ribs demand strapping or the diachylon bandage. Pneumothorax, artificial or traumatic, often arrests severe pulmonary bleeding by contracture of the lung tissues upon the bleeding vessels. If hemorrhage continues, the pleural sac is widely opened and the ligature or tamponade is employed. Marked dyspnea calls for aspiration of the pleural contents. The treatment of infection is taken up on p. 1831. Rupture of the diaphragm, with or without hemorrhage, should be repaired by thoracotomy (p. 1848).

Gask[4] says "When a missile has traversed both the chest and abdomen, the diaphragm is necessarily injured and abdominal viscera may invade the pleural cavity. This is more common on the left side than on the right, as the latter is protected by the liver. Employment of the transpleural route in conjunction with the abdominal is at times necessary in order to repair the diaphragmatic rent. The war has modified and perfected the technic of acute thoracic surgery and demonstrated the fallacy of the expectant plan of treatment. It has also emphasized the fact that a special differential pressure operating cabinet is not necessary in this class of cases." A special classification and outline of treatment in gunshot wounds is quoted from the report of Lilienthal,[1] as follows:

"*Sucking wounds* are those in which there is a free access of air to and from the interior of the chest. These wounds, when small, are not necessarily dangerous. Traumatic openings, however, when large enough to admit air by suction, in quantities as great as, or greater than, that which enters the lung through the larynx, give rise to mediastinal flutter with its attendant dangers. In these cases it is best to close the chest by simple skin suture or by firmly covering

the wound with wet gauze. If the wound is very large, the patient's distress, due to the mediastinal motion, may be instantly relieved by traction on the presenting lung (p. 1818). This steadies the mediastinum and the lung may be kept in contact with the chest wall by a few sutures or by a sterile safety pin. No great traction force is necessary. In from twenty-four to forty-eight hours the pleura covering the mediastinum is stiffened by exudate, thus obviating the danger of 'flutter.' The presence of the exudate makes opening of the infected chest less dangerous than wounding of the normal thorax. Thoracotomy for empyema illustrates this relative immunity."

Large sucking wounds presuppose gross injury to the lung and other structures, besides the probable implantation of infected materials, such as clothing. Fifteen of these cases were treated by Lilienthal[1] with six fatalities — a mortality of 40 per cent.

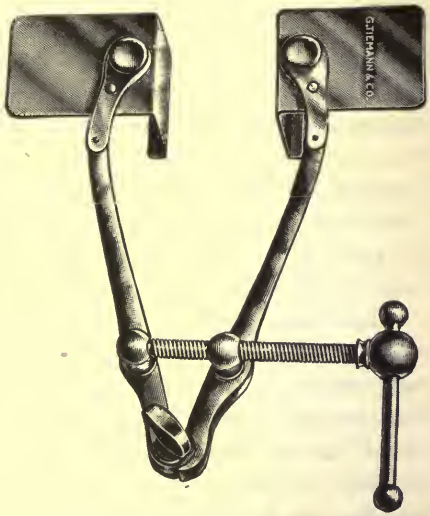

Fig. 851.— Lillienthal's Rib Spreader.

Penetrating wounds include those in which the missile is within the chest or in the thoracic wall, having passed completely through one wall of the cavity, but with no wound of exit. Out of thirty of this class there were nine deaths — a mortality of 30 per cent.

Of *completely perforating wounds* with entrance and exit (no foreign body retained), out of twenty-one cases, two died.

Lilienthal's summary of conclusions is as follows: "The wounds seen in evacuation hospitals may be divided in two classes: (1) Those which demand repair of obvious injuries to the chest wall, such as large open wounds or extensive fractures of the ribs with probable injury to the lung. (2) Injuries to the lung or large foreign bodies in the thorax which demand exploration. Wounds in class 1 should be treated on ordinary surgical principles and the wound closed as well as possible. In the presence of large defects in the pleura obliteration may be accomplished by suturing the inflated lung to it. When this is not feasible, flaps may be used for the purpose.

Wounds in class 2 should be operated upon by what we have denominated as major intercostal thoracotomy without resection of ribs. With the aid of a well constructed rib retractor (Fig. 851) an ample opening can be made, through which all parts may be clearly visualized and through which operations on the lungs, diaphragm, and most parts of the chest wall can be performed with comfort to the operator. In concluding the operation, the ribs may be easily brought together and the pleura fully approximated by three pericostal sutures of absorbable material (kangaroo tendon or chromicized catgut). Whether or not there should be approximation of the skin is a matter to be decided in each case.

"There may be combinations of class (1) and (2) by extending an already large wound in any part of the chest. The principle of visual exploration with the aid of a rib spreader can be applied here.

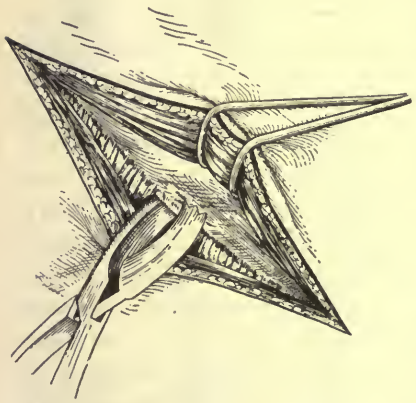

FIG. 852.— CONTROL OF BLEEDING VESSEL AT LOWER EDGE OF RIB.

"Some kind of forced anesthesia, such as the intrapharyngeal method already referred to, is absolutely necessary in treating recent wounds of the thorax by full exploration.

"All thoracic wounds were kept under observation for not less than ten days at the evacuation hospital, and even then, we were not sure that complications — mechanical or through infection — might not occur at a later period.

"Pneumonitis of the opposite lung is the greatest immediate danger in these cases. While its cause was unknown, the patients were guarded against cold and exposure, especially during the change of dressings."

Duval[3] states "My investigations show clearly that a gunshot wound of the lung presents exactly the same lesions as does any other war wound and is subject to the same evolution, 1. operations of urgency — (a) serious external hemorrhage; (b) open thorax and mechanical difficulties in respiration; 2. early operations for wounds of the lung.

"1(a). *Serious external or internal hemorrhage* calls for immediate thoracotomy; suture of the bleeding wound of the lung or ligature

of the bleeding point. If the general condition of the wounded man allows, the complete surgical treatment of the wound of the lung and of the parietal wound, and complete closure of the chest after cleansing of the pleural cavity, are executed.

"1(b). In *open thorax* some surgeons are content to suture the skin wound without treating the injury of the chest cavity. Statistics show that this method of combating asphyxia is good, but it does not protect the patient from those complications which follow the normal evolution of the lung wound, and the mortality of open chests treated by suture alone is about 20 to 24 per cent. In our opinion it is not logical to close the thoracic cavity without treating the wound of the lung and, even when the condition of the patient is serious, it is best to perform the complete operation.

"*In early operations for wounds of the lungs,* apart from those of urgency, the indicadtions for operation are difficult to define. The following, however, may be cited: All foreign bodies retained in the lung, unless they are very small, should be removed. All wounds of the lung, complicated by a fracture of the ribs, should be operated upon, because they contain splinters of bone (intrapulmonary splinters are visible only by Röntgenography, and not by the fluoroscopic screen). Every wound of the lung which, on fluoroscopic examination, shows a large intrapulmonary hematoma should be operated upon, because this hematoma almost always becomes infected. Even tangential wounds of the thorax with pulmonary lesion should be submitted to the same treatment. Apart from these cases, through and through wounds by rifle bullet without serious hemorrhage, and wounds containing very small fragments should not be operated upon. It is important to remember, however, that the comparative freedom from danger in surgical intervention renders the indication for operation more and more frequent.

"According to our latest statistics for operations not of urgency, we have operated upon eighteen cases out of one hundred and eighteen. The favorable time for operation upon the lung is as soon after injury as possible. After thirty hours it is, as a rule, advisable not to perform any operation.

"In operations of urgency in cases with hemorrhage and open thorax, we have cured 66 per cent of our wounded. In thirty-five prophylactic operations for infection of the lung and pleural cavity, there were no deaths."

Bastianelli[2] advises the production of an artificial pneumothorax

in lung injury in the majority of cases and even after other operative procedures have been performed. He advocates the use of the Morelli bags (Fig. 853) for occluding open wounds and the Morelli apparatus for production of artificial pneumothorax (p. 1847).

Gask,[4] in speaking of types of wounds, says "The types of wounds which are most commonly met with in the casualty stations are (1) through and through wounds caused by rifle bullets; (2) through and through wounds caused by shell fragments; (3) lodging wounds with retention of large foreign bodies; (4) lodging wounds with retention of small foreign bodies; (5) open sucking wounds of the thorax with or without retention of foreign bodies.

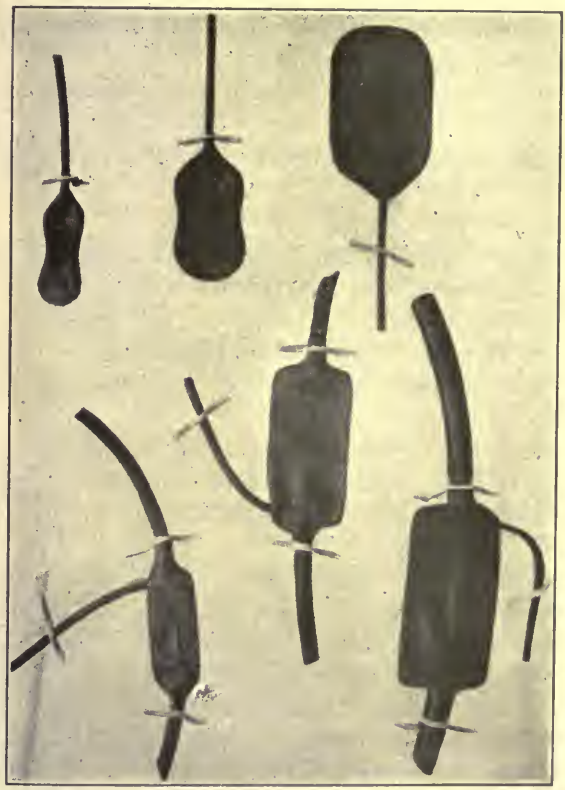

FIG. 853.— THE UPPER RUBBER BAGS ARE USED TO PLUG A CHEST OPENING. THE LOWER RUBBER BAGS, WITH INTERIOR DRAIN ARE FOR SUCTION TREATMENT OF EMPYEMA (Morelli).

"Indications for early operation are: a ragged wound of the soft parts; bleeding from the parietal wound; compound fracture of the ribs; suction of air into the pleural cavity; retention within the chest of a large foreign body; great pain due to indriven splinters of bone; rapidly increasing pneumothorax, due to a valve-like opening in the pleural cavity which allows air to be sucked in and prevents expulsion; and a large hemothorax which cannot be evacuated by aspiration. When none of these indications is present, that is, when the wounds of the chest wall are small and clean, such as

are made by a rifle bullet, when the ribs are not splintered, and when the foreign body retained is small, the patient is treated on general medical principles."

Turner[5] says in his summary "The indications for operation in *late cases,* are as follows:

"1. Foreign bodies in the parieties with, or without, sinuses.

"2. Foreign bodies in the lung, irrespective of size, if associated with persistent cough, hemorrhage or suppuration.

"3. Large foreign bodies in the lung, even if the symptoms are purely nervous.

"4. All foreign bodies lodged in the pleura with, or without empyema.

"5. All cases of infected hemothorax.

"6. All cases of through and through wounds with shrinking of the side, lessened lung expansion, and interference with movements of the diaphragm in which treatment by exercises, etc., has failed.

Surgical Diseases of the Pleura.— Injury to the pleura has been discussed in conjunction with penetrating and crushing wounds of the chest and pneumothorax.

Air may enter the pleural cavity from penetrating wounds of the chest, lung perforations, trauma, rupture of a tuberculous cavity and in connection with infection with aërogenes capsulatus in the pleural exudate.

Hydrothorax is a transudation. The fluid is a clear straw color and does not contain fibrin or bacteria. It appears as a part of general anasarca which may be due to cardiac decompensation, renal insufficiency or hemic conditions. It becomes surgical when caused by inflammatory diseases of the pleura, by new growths or trauma, in accord with which the fluid takes on different characteristics depending upon whether it is due to hemorrhage or infection. In these instances the condition of the fluid changes and, strictly speaking, they should be termed hemothorax or pyothorax, as the case may be. The effusion is usually bilateral, diminishing the thoracic cavity and causing displacement of the lung and heart. According to Stengel,[6] unilateral effusion usually follows cardiac disease. The hydrothorax in these cases is, as a rule, right sided and is probably due to pressure upon the azygos veins and root of the right lung.

Dyspnea, if associated with cardiac or renal disease, arouses suspicion of the effusion. The physical signs depend upon the amount of effusion. *Inspection* reveals bulging of the intercostal spaces; *pal-*

pation, absence of fremitus; *percussion,* flatness; and *auscultation,* absence of respiratory sounds and vocal fremitus.

The *surgical treatment* consists in *aspiration* of the fluid. This must often be repeated. Permanent drainage is rarely necessary.

Hemothorax designates free blood in the pleural cavity. It is caused by wounds of the chest, rupture of an aneurism, and new growths of the pleura. In the last condition the effusion is hemorrhagic, but may be serous at first. In the absence of *infection* the blood is usually absorbed. In *traumatic* cases the onset may be insidious, but one should be alert for signs of hemorrhage. In ruptured aneurism, the hemorrhage is fatal. The signs are those of fluid in the chest plus those of hemorrhage, the degree depending upon the amount of bleeding.

The *prognosis* depends upon the cause. In traumatic cases, the size of the vessel, its location and the possibility of ligature of the bleeding vessel (if spontaneous clotting does not occur) are deciding factors.

The *treatment* consists in absolute rest in bed, morphin (or pantopin), chest strapping or diachylon bandage, ice bag. etc. Arrest of the bleeding by ligation, cautery, or packing is indicated. See Gunshot Wounds, etc., p. 919.

Chylothorax may be defined as a collection of chyle in the thoracic cavity. It is due to injury to the thoracic duct, especially in gunshot wounds. Several cases have been reported following suppurative or malignant disease. The chyle empties into the mediastinum from the pleural cavities when a communication exists between them. Chylothorax usually escapes recognition in traumatic cases, the other visceral injuries and hemorrhage overshadowing the injury to the duct. If the duct involvement is the important injury, gradual accumulation of fluid in the chest cavity, and progressive loss of weight and digestive disturbances are the important symptoms. A positive *diagnosis* can be made only by aspiration.

If the duct does not heal spontaneously, the *prognosis* is unfavorable, as operative measures do not afford relief. Repeated aspirations have been followed by recovery in a few cases. The outcome depends, to a large degree, on the size and character of the injury to the chest.

Pyothorax is an accumulation of pus in the pleural cavity. It is produced from without by infected penetrating wounds; from within, by pneumonitis, abscess of the lung, mediastinal or subphrenic suppuration, and also by an infected focus in the chest wall, and may be hematogenous in origin. In most cases of empyema the exudate is at first serous, and, later, purulent, in character. In penetrating

wounds, however, the fluid is purulent from the beginning. The serous fluid is straw or amber colored, after a time becoming cloudy, and finally opaque. The organism responsible for the infection has some bearing on the *color and consistency of the fluid.* The pneumococcus produces a thick, creamy, yellow or greenish pus; the streptococcus, a watery and flocculent pus; the tuberculous variety, a thin fluid containing numbers of caseous masses. The odor is at times nauseatingly sweet; following external wounds it is fetid. The colon bacilli produce a foul-smelling pus, while cases secondary to gangrene of the lung have a putrid odor. The fluid usually separates into a clear upper, and a creamy lower, layer.

The *pleural surfaces* are greatly thickened and are more or less covered by a fibrinous exudate. If accumulation of pus is limited by adhesions, a sacculated empyema is developed. This may be interlobar or located between the diaphragm and the base of the lung.

Empyema may terminate by absorption of the fluid (if the amount is small); by rupture into the esophagus or stomach; by external rupture, through an intercostal space; or it may erode a bronchus, causing immediate or subsequent suffocation; or, like a tuberculous abscess of the spine, point at the groin. Finally death occurs by exhaustion from toxemia and embarrassed respiration.

The *symptoms* may develop insidiously or the onset may be sudden. They may be those of a simple effusion with compression, to which are later added those of septicemia, or those of sudden compression with marked toxemia. With an insidious onset, the clinical picture may simulate a pulmonary or abdominal condition; and embarrassed respiration, intercostal bulge, and irregular remission of temperature may not occur until later. In some cases chills and sweats are the initial symptoms. *Leukocytosis* depends upon the type of organism. The physical signs are those discussed in simple effusion. The disproportion in the two sides of the chest, together with the intercostal bulge, is especially noticeable in children.

In clearly defined cases the *diagnosis* is easy. When the onset is insidious the condition may be overlooked. *Aspiration* is often necessary before a positive diagnosis can be made. The X-ray is of great value in localization, especialy in the sacculated variety.

The *prognsois* varies with the type of causative organism; in pneumococcus empyema it is generally good. The toxemia accompanying streptococcus and staphylococcus infections makes the outlook unfavorable; this is also true with regard to the tuberculous type of the

affliction. Empyema following rupture of a subphrenic abscess and gangrene, or abscess of the lung, is usually preceded by a period of exhaustion which renders the outcome unfavorable.

The *treatment,* except in the tuberculous variety, is operative. This should be executed as soon as the diagnosis is made. The exact location of the exudate should be verified with the aspirating needle immediately before the thorax is opened. Aspiration in certain cases of pneumococcus empyema, in which the pus is sterile, may be followed by relief.

Mozingo[7] uses what he calls the closed method with Dakin's solution. For the purpose the apparatus of Morelli (p. 1847) may be used.

Mozingo[7] makes the following statements:

"1. Early operation by the closed treatment method has the following advantages:
   - "(a) It can be done without shock to the patient or collapse of the lung.
   - "(b) It provides perfect evacuation and cleansing of the empyemic cavity.
   - "(c) It prevents absorption of toxins.
   - "(d) It prevents the lung, compressed by the exudate, from becoming fixed in compression.

"2. Sterilization is more rapid by the closed, than by the usual, methods, as the antiseptics are held in contact with the infected surfaces until they have exerted their full bactericidal action.

"3. Dakin's solution followed by a two per cent dilution of liquor formaldehyd in glycerin is the most ideal antiseptic method of treating empyema.

"4. Communication of an empyemic cavity with a bronchus is more common than is generally suspected. It is not a contra-indication to the use of Dakin's solution and formaldehyd, and closes more readily when these are used.

"5. Constant negative pressure gives the maximum expansion of the lung, and the irritating and cleansing action of sodium hypochlorite solution causes rapid obliteration of the cavity.

"6. In uncomplicated cases of empyema the patient need not be kept in bed.

"7. Test smears are negative, although cultures are positive when Dakin's solution is used, and vice versa when formaldehyd-glycerin is employed.

"8. Röntgenographic and bacteriologic laboratory facilities, while always to be preferred, are not absolutely essential in treating empyema by the closed method.

"9. The closed method is productive of great economy of time, labor and dressing material.

"10. Treatment by the closed method can effect cures in many cases similar to those that are usually treated by the Schede, Eastlander or Delorme operations.

"11. The closed method is practicable in the home and country practice.

"12. A hypochlorite solution of increased strength over Dakin's solution, in both alkalinity and available amount of chlorin, can be used in empyemic cavities with safety."

The writer feels that Mozingo "preaches a heresy." The conception of the method cannot be based on the diverse pathological conditions present in cases of empyema. However, experience may prove the contrary.

The technic of thoracotomy is described farther on (p. 1849). After the pleura is opened the exudate is *gradually* allowed to escape, the rate of outflow being controlled with a wet pad. The condition of the cavity may then be explored with the gloved finger.

A drainage tube is inserted and Bryant's[8] "bull dog" — a postoperative aspiration apparatus — may be attached to the drainage tube, or a simple double tubed bottle may be connected with it. The dressing should be changed frequently. Irrigation has its limitations. It may be used, however, when drainage is obstructed by necrotic tissue. A two per cent liquor formaldehyd in glycerin may be employed for the purpose. Dakin's solution has recently become *en vogue* and good results have been reported; Dakin's in combination with the former has also produced good results.

In early cases removal of pus is followed by immediate expansion of the lung. Later, when organization has taken place, the expansion may be aided by having the patient blow on a trumpet or a bugle. This method has come into prominence during the war. A good substitute is making soap bubbles with an ordinary clay pipe, or using Wolff's bottles (Fig. 854), where fluid is transferred from one bottle to another by blowing. In early cases Bryant's[8] "bull dog" works exceptionally well. In late cases, where fibrinous organization has occurred and the collapsed lung is bound down by a thick fibrinous exudate, Fowler[9] and Delorme[10] advise opening the thoracic cavity freely

by an osteoplastic flap and removing fibrinous exudate from the visceral layer of the pleura.

An immediate partial, and later complete, expansion of the lung may occur. If this fibrinous exudate is not removed, the lung undergoes a cirrhotic change which prevents expansion and the suppurative pleural cavity persists. As the lung cannot expand, several operations have been devised to approximate the chest wall to the lung.

FIG. 854.— WOLFF'S BOTTLES.

(From Keen's Surgery, copyright by W. B. Saunders Co.)

Estländer's[11] *operation* is performed in cases where the cavity is small. It consists in removing several ribs, leaving the periosteum and soft parts intact, and allowing the collapse of these parts, thus obliterating the hollow space. In the larger type of cavity this method is inadeqate, owing to the stability of the intercostal muscles and periosteum (p. 1852).

Schede's[12] *operation*, which is largely practiced, consists in the

complete removal of ribs and intercostal muscles, leaving a flap of skin, fascia, and superficial thoracic muscles, which is sutured in place and made to lie in contact with the collapsed lung (p. 1852).

The *surgical treatment of tuberculous empyema* is a debatable question. As the pus rarely gives rise to severe toxemia, and mixed infection, with its toxic syndrome, almost always follows the operation, it is best to withhold interference until the symptoms become menacing. In this event, asepsis must be carefully practiced. *Aspiration,* and, later, injection of emulsion of iodoform is the method of choice.

New Growths of the Pleura.—Primary growths of the pleura are very rare and are of mesoblastic origin. The *secondary growths* are more frequent and follow direct extension from the lung, the mediastinum or chest wall. They are also metastatic in origin.

Primary Growths of the Pleura.— Endothelioma appears as a diffuse growth or in the form of nodules. Effusion may occur and is likely to be, though not necessarily, tinged with blood. The tumors develop insidiously, and, when symptoms are present, simulate pleurisy with effusion; pain, when present, is no different from that accompanying dry pleurisy. The *diagnosis* is made by aspiration and resistance of tissue. The physical signs remain practically the same after the fluid is removed. Blood tinged fluid is indicative of new growth.

Sarcoma of the pleura simulates endothelioma, being single, multiple and diffuse. As elsewhere in the body, its growth is rapid and bloody fluid is more likely to be present than in endothelioma. Rapid afebrile accumulation of fluid without apparent cause is of diagnostic value. X-ray is helpful, though it does not differentiate lung growths. Microscopical examination of the aspirated fluid may lead to a conclusion, especially when it contains mitotic figures.

Secondary Growths.— Carcinoma and sarcoma occur more frequently and give practically the same signs and symptoms as primary growths. The diagnosis in this class of cases is less difficult when the primary growth is recognized elsewhere. The nature of the growth and its location usually limits the treatment to aspiration, X-ray and radium. In secondary growths which spread by direct extension from the chest wall, surgical extirpation is possible.

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CHAPTER IV

SURGERY OF THE LUNG

Injuries of the lung have been taken up in connection with those of the chest wall (p. 1816).

Surgical Diseases of the Lung.— *Abscess of the lung* is comparatively rare. Its etiological factors are numerous. It occurs most often as a sequel to bronchopneumonic infections. It may follow the lodgment of foreign bodies in a bronchus, hemorrhagic infarcts with infection, perforating empyema, infected penetrating wounds, and it may be hematogenous. The abscess, as a rule, is single, the invading organism depending upon the primary cause, i. e., secondary to pneumonitis (pneumococcus), secondary to pyogenic infection (staphylococcus, streptococcus), etc. Mixed infection is the rule.

The sudden onset of the *symptoms* of fever, following pneumonitis, without additional signs of lung involvement should arouse suspicion of abscess. This is made certain by the expectoration of a large amount of foul smelling pus. When the cause is other than pneumonitis, the history of the case must be taken into account. If rupture into a bronchus is delayed, the physical signs are those of the first stage of pneumonitis. After rupture, physical signs of cavitation appear, with sibilant and sonorous râles and amphoric breathing. Localization by physical signs is often misleading, especially if the abscess is of the central type. The X-ray findings are helpful. *Exploratory aspiration* should be employed only if the surgeon is prepared to operate at once.

The *prognosis* of abscess, if drained without infecting the pleural cavity, is, as a rule, favorable.

When the abscess is located it is exposed by thoracotomy. The presence of pus is determined with a director and the opening enlarged with blunt scissors or an artery clamp. If the process is attacked before adhesions are formed, this may be overcome by Keen's[1] method which he describes as follows: "A rectangular flap is made by an incision 5 to 6 cm. long in the axis of a rib and two short vertical

incisions at each end. This exposes one, or, if the lateral incisions are made sufficiently long, two intercostal spaces. By light touches of the knife the intercostal muscles are gradually divided and pushed up and down until the parietal pleura is reached. If there are no adhesions, the mottled surface of the lung can be seen moving up and down with each respiration. If adhesions exist, the pleura is opaque and no such motion is seen. In order to produce adhesions it is necessary only to pack the wound for three or four days with iodoform gauze and hold the flap in place by a few temporary sutures. The abscess can then be opened without danger of infecting the pleura."

When *rupture into a bronchus* occurs, the question of external drainage is a debatable one. In a certain number of cases healing occurs spontaneously. Delay in healing is due to the formation of strong adhesions of the lung to the chest wall so as to prevent collapse of the pus cavity after spontaneous drainage is complete. Murphy[2] advocates production of a pneumothorax with nitrogen gas when no adhesions are present, and thoracotomy and breaking down of adhesions, if any be present. He determines this point as follows: a hypodermic needle is introduced over the site of the abscess and, when the point is embedded in the intercostal muscle, a rubber tube is attached which connects with a reservoir of sterile nitrogen gas; the needle is then pushed slowly inward. When the needle enters the pleural cavity, the gas passes rapidly through it and produces an artificial pneumothorax. If adhesions exist, no gas will enter through the needle.

Gangrene of the Lung.— Gangrene of the lung is closely allied to abscess. Like abscess the predisposing etiological factors are asthenia, diabetes, nephritis and alcoholism. The most frequent exciting cause is pneumonitis, to which may be added ulcerative bronchiectasis by extension of a putrefactive process, and embolism of a branch of the pulmonary artery which forms an hemorrhagic infarct.

The *pathological picture* is that of an area of necrotic lung tissue infected by putrefactive bacteria. The gross appearance of the lesion is that of a dark green or grayish slough separated from healthy tissue by a sharp line of demarcation. The process may involve an entire lung.

As in abscess of the lung, the *symptoms* are first those of the predisposing cause, followed by an indescribable stench of the breath. The odor, as compared with that of abscess of the lung, is much more fetid. This is followed by cough and abundant expectoration of foul smelling secretion. Shreds of necrotic lung tissue may be expelled. The

temperature is not high and may be subnormal. The general picture is that of extreme prostration with feeble pulse and rapid shallow respirations. Hemoptysis occurs, if one or more vessels ulcerate. This is rarely fatal.

The *diagnosis* is not difficult. Localization of the lesion and the question of its multiplicity is not easily determined. The X-ray is of great value.

The *treatment*, until the latter part of the nineteenth century, was purely medicinal. The death rate was as high as 80 per cent. *Operative measures* have reduced this to 30 or 40 per cent. Bull[3] of Copenhagen in 1881 operated for the first time successfully upon a case of gangrene of the lung. Only a circumscribed single focus is amenable to surgical attack. The procedure is similar to that employed in connection with abscess of the lung, the avoidance of infecting the pleura being equally imperative. One or more ribs must be resected. Obliteration of the pleural cavity, if there are no adhesions, is accomplished by suturing the two layers of the pleura together. Unless the sloughing lung area has separated, no attempt should be made to remove it. The lung cavity is packed with gauze and the external wound kept open until the slough separates. Healing takes place by granulation.

Bronchiectasis.— Bronchiectasis may be defined as a dilatation of a bronchus of which there are two main types, the *cylindrical* and the *sacculated*. The process may be *general*, or *localized*. It is rarely congenital and usually follows interstitial pneumonitis. The weakening of the bronchial wall is the causative factor. The cylindrical and fusiform dilatations are usually bilateral. As a rule the sacculated variety is unilateral. The chief underlying causative factors are adhesions, chronic cough (bronchitis-pneumonitis), tuberculosis, aneurism, tumors, foreign bodies and lues. After dilatation has occurred, the secretions are retained and later become infected. As the cavity is distended, overflow produces cough which empties the sac so that the patient is relieved until the cavity fills again. Only the unilateral, localized type is amendable to surgical relief.

The *symptoms* are chronic paroxysmal cough and profuse expectoration of foul smelling pus; these are characteristic of the sacculated type. Cough usually occurs in the morning and is attended with gradually increasing prostration and fever. Physical signs vary with the character of the contents of the cavity. Large moist râles are present when the saccules are full and disappear when they are

empty. X-ray is of value. Aspiration should be performed only when the surgeon is prepared to operate immediately.

The *prognosis* depends upon the extent of the sacculations and upon the primary causative factor. The condition may exist for years. Death is usually the outcome of gradual exhaustion. Surgical efforts at relief are permissible when the lesion is unilateral and localized. This is, however, difficult to determine. Although multiple sacculations have been successfully operated on, the mortality is high.

The *surgical treatment* is similar to that employed in connection with abscess.

Tuberculosis of the Lung.— Tuberculosis of the lung as a surgical problem is enmeshed in the consideration that a single lesion is comparatively rare. In the stage of cavitation, surgical measures have not been sufficiently rewarded by success to warrant continuance of the effort. Although patients frequently survive the operation, few are permanently relieved. In cases of severe toxemia (the result of mixed pyogenic infection) drainage may be justified. In cases where cavities exist and do not respond to proper medicinal treatment, and the rigidity of the thoracic wall prevents healing, artificial pneumothorax, to produce lung collapse and subsequent cicatrization, is practiced by Murphy.[4] Various methods have been devised for the purpose, including removal of ribs over the cavity to allow collapse of the soft parts (Garré,[5] Quincke,[6] Landerer[7]). Murphy's[4] method of production of pneumothorax by sterile nitrogen gas is employed in the absence of adhesions. It is followed by shrinkage of the cavity and quiescence of the lung and is attended with a very low mortality.

Actinomycosis of the Lung.— Actinomycosis of the lung is rare. When it is caused by inhalation of the ray fungus (streptothrix, actinomyces), a primary focus is developed in the lung. A *secondary lesion,* following one in the abdomen, is much more common. The condition simulates tuberculosis and in the beginning produces little systemic reaction. Later, cavitation occurs. The excessive amount of granulation tissue with subsequent contracture often gives rise to depression of the thorax. The pleura may become involved by direct extension, and finally the soft parts, the subcutaneous tissue, the mediastinum and the abdomen are invaded.

Because of the dense pleural adhesions, effusion into the thoracic cavity is limited.

Symptoms, in the incipient stage of the disease, may be absent or may be those of bronchitis or of incipient tuberculosis. Later, pain,

cough, fever, general debility and loss of weight are added to the clinical picture. Expectoration is scant at first, and not characteristic. If rupture into a bronchus takes place, expectoration is more abundant. Hemoptysis may occur. The absence of tubercle bacilli and the presence of actinomyces make the *diagnosis* positive.

The *prognosis* is grave.

The *surgical treatment* is practically the same as that of incipient tuberculosis. Schlange[8] reports three recoveries. When suppuration takes place, drainage of pus cavities may be practiced.

New Growths of the Lung.— New growths of the lung are *benign and malignant* in character, the latter being *primary* or *secondary*. Almost all varieties of benign tumors have been found in the lung. They rarely attain sufficient size to produce symptoms and do not demand surgical aid. The echinococcus cyst is the exception.

Echinococcus cyst of the lung is second in frequency to that of the liver. The condition is rare in the United States. Its danger lies in displacement of thoracic viscera and in occasional rupture into a bronchus. Secondary infection of the cyst may occur. The symptoms are those of gradually increasing intrathoracic pressure, dyspnea, afebrile cough, and, later, bloody expectoration. Late in the disease bulging of the chest wall and displacement of abdominal viscera occurs. The *diagnosis* is made on the history of tumor, the finding of hooklets in the aspirated fluid, with a positive echinococcus complement fixation test. X-ray is of value in establishing the presence of encysted fluid. The *prognosis* is grave. Death may result from rupture into a bronchus or from secondary infection.

In the *treatment,* thoracotomy, and suture of the sac to the external wound to produce continuous drainage, is indicated. Occasionally the secreting layer of the sac may be removed. V. Eiselsberg[9] has succeeded in completely enucleating a cyst. Because of the danger of pleural infection, aspiration should be immediately followed by operation.

Malignant tumors, with reference to their treatment and diagnosis, are considered together.

Endotheliomata of the lung are secondary to pleural involvement and are described under new growths of the pleura.

Carcinoma occurs as a *primary,* and as a *secondary* growth. The former is rather rare. It has its origin in the epithelial cells of the bronchial mucous glands or in those of the alveoli. It may occur in the form of numerous nodules or an entire lobe may be involved.

Sarcoma occurs as a primary growth and is less frequent than car-

cinoma. It develops as a lymphosarcoma, which grows along the bronchial tree and later infiltrates large areas of the lung. It rarely arises primarily from lung tissue. The spindle cell sarcoma, as a rule, is confined to one part. Both secondary carcinoma and sarcoma are much more frequently observed than are the primary growths. As a rule they are multiple.

In *primary growths* the condition is rarely suspected until late in the process, when afebrile cough, with physical signs of consolidation and no râles, arouses suspicion. Later, pain and slight dyspnea, and still later, bloody expectoration occur. At the same time aspiration reveals the presence of bloody serum, and *cachexia* and supraclavicular enlargement of lymph nodes develop. Signs of compression, manifested by atelectasis or bronchial stenosis may occur. Pneumonitis is a frequent complication of the condition. Age is of some diagnostic importance. Sarcoma may develop at any age, most frequently in middle life, while carcinoma usually appears late in middle life.

Secondary Growths.— In secondary growths the lung symptoms develop in the course of the clinical picture presented by the primary focus. Those referable to the pulmonary involvement are similar to those described in connection with primary growths.

The *diagnosis* is based on the symptoms as stated, the character of the aspirated fluid, the physical signs, and the Röntgenographic findings. As a rule the diagnosis in primary growth is not made until cachexia develops. However, with regard to the aspirated fluid, Lenhartz[10] considers the admixture of large and numerous fatty globules as pathognomonic. E. Fraenkel[11] maintains that the presence of very large vacuole cells — ten to twenty times the size of leukocytes — is indicative of the presence of sarcoma.

In *secondary growths*, the presence of the primary lesion and the development of an afebrile cough, the signs of consolidation, without the clinical picture of inflammation, are indicative of metastatic growth in the lung.

Pneumectomy is justified, if the diagnosis is made early enough.

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CHAPTER V

THE SURGERY OF THE MEDIASTINUM

Injuries to the Mediastinum.— Injuries to the mediastinum are caused by accidents of a crushing nature, such as gunshot and stab wounds, and by fracture of the spine or sternum. *Involvement of the heart and blood vessels* is described in connection with injuries to the heart and great vessels. *Hemorrhage into the mediastinum* from smaller vessels is rarely of serious consequence, as it is small in amount and distributes itself easily throughout the cellular tissue. It is readily absorbed if infection does not ensue. Pressure symptoms of an alarming nature are seldom produced.

Injury to the trachea or to the bronchi gives rise to emphysema, which gradually fills the cellular tissue of the mediastinum and eventually appears at the root of the neck, finally spreading extensively throughout the subcutaneous tissues of the body.

The *diagnosis* is easy and is based on a puffing and distention of the tissues, which give a soft, crackling, crepitus on pressure.

Mediastinitis.— Acute mediastinitis usually manifests itself in the form of an abscess. Hare[1] collected 115 cases of mediastinal abscess, of which 44 were acute. The abscess is usually situated in the anterior mediastinum. The *causative factors* are as follows, infection of an external wound with injury to the trachea, to a bronchus, or to the esophagus, or perforation of these structures from ulceration produced by foreign bodies; deep cervical cellulitis by direct extension from inflammatory conditions of the lung, pleura, and osseous structures.

Tuberculosis is the most frequent cause of the chronic variety, the abscess occurring secondarily to caries of the spine or to tuberculous lymph node suppurations.

Pain behind the sternum is the *chief symptom.* It is deep seated, localized, throbbing and may become very severe. It radiates in various directions and is associated with fever, chills, sweats, general malaise, loss of weight and hemoptysis.

Large abscesses produce pressure dyspnea. The pus may burrow

into the abdominal cavity or point at the root of the neck, perforate an intercostal space or erode the sternum.

The *physical signs* are not definite. Tenderness, at times, helps to localize the abscess. A pulsating and fluctuating tumor may occur at the border of the sternum or at the sternal notch. The absence of a bruit and of expansive pulsation differentiates the process from an aneurism. When there is doubt, aspiration with a fine needle is permissible. In chronic abscess the constitutional symptoms are usually absent. The *prognosis* is grave in the septic type, especially when the original infection has already reduced the patient's general condition. In the chronic type the prognosis is less unfavorable. Rupture into the trachea causes suffocation. The *treatment* consists in *absolute* rest in bed, ice bag to the chest and supportive medication. If the abscess causes bulging of the soft parts, it should be incised and drained. If localization of the process is limited to the anterior mediastinum without pointing in the soft parts, the sternum may be opened and drainage employed. The posterior mediastinum is approached by an incision over the transverse processes, several of which, together with a portion of the ribs, are resected. This is a very difficult procedure (p. 1854).

Tumors of the Mediastinum.— In comparison with the malignant, benign tumors of the mediastinum are very rare. However, neither class of growth is frequently met with.

Benign Tumors.— Of the *benign tumors, lymphomata* are the most frequent, with *dermoid cysts next. Lipomata, gummata, enchondromata,* and *echinococcus cysts* also, occasionally develop in this situation (Hare[1]). The anterior mediastinum is more frequently invaded than the posterior. Only the dermoid and the echinococcus cysts give rise to disturbances.

Malignant Tumors.— Of the *malignant growths,* carcinoma most frequently develops in the mediastinum. Carcinoma is usually secondary, while sarcoma is likely to appear primarily in this situation. The former develops, as a rule, from remnants of the mucous membrane of the esophagus or trachea displaced during embryonic evolution. Sarcoma usually appears in the lymphosarcomatous form.

The *symptoms* and signs are the outcome of intrathoracic pressure. The onset depends upon the rapidity of increase in the size of the growth. In *benign growths* the thoracic vicera compensate by adapting themselves to the increased pressure. On the other hand, rapidly growing malignant tumors cause early pressure symptoms, and some-

times death occurs before cachexia develops. Dyspnea is one of the earliest and most constant symptoms and is due to one of the following causes, pressure on the trachea or recurrent laryngeal nerve, pressure on the heart or great vessels, and pleural effusion. Cough is usually a concomitant symptom with dyspnea. It is paroxysmal and of a "brassy" quality, if the recurrent nerve is involved. The voice may also be affected by the same cause. Pain is a reliable symptom; it is paroxysmal and radiating in character. Pressure on the vessels is common. The veins are the more compressed and may be obliterated; this results in cyanosis of the face and upper extremity, depending upon the vein involved. Pressure on the esophagus produces dysphagia. At times, pupillary changes are produced by pressure on the sympathetic. Unilateral and bilateral edema of the chest is produced by compression of the vena azygos. Cases have been reported in which this sign was the only one present. Dysphagia and emaciation from pressure on the thoracic duct occur late in posterior mediastinal growths. Expectoration of blood and pus is characteristic of dermoid cyst. Pleural effusion, when it occurs, changes the physical signs.

When the *diagnosis* of thoracic growth has been established, the following helps to localize the tumor: a growth located in the anterior mediastinum pushes the sternum forward and often erodes it; the growth may be palpable in the suprasternal notch; the cervical lymph glands are usually involved: dyspnea is very common; and the pressure is generally on the venous trunks. In the middle and posterior mediastinum the tumors produce severe dyspnea and cough, and the esophagus, the recurrent laryngeal nerves, the vena azygos, and the thoracic duct bear the brunt of pressure.

The differential diagnosis must be made from aneurism — a difficult problem. The Wassermann test, history of lues, the duration of the condition and "diastolic shock" are indicative of aneurism. Pressure symptoms are almost identical in aneurism and in thoracic growth.

Operative removal of a new growth in this situation is justified when the diagnosis is made early.

The surgery of the thymus gland is taken up elsewhere (Part XIII, chap. i).

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CHAPTER VI

OPERATIONS ON THE THORAX

Paracentesis of the Thoracic Cavity.— Paracentesis of the thoracic cavity is employed for the therapeutic removal of fluid exudate and air for diagnostic purposes. The patient may be seated with the head and shoulders bent forward, or may lie in bed, on the sound side, with a sand bag under the chest. Unless the fluid is encapsulated, the aspirating needle is inserted in the midaxillary line or on a line with the angle of the scapula in the sixth or seventh intercostal space. The arm should be raised and the skin drawn slightly upward. The needle is thrust inward and upward, hugging the upper border of the rib. Entry into the pleural cavity is announced by a sudden absence of resistance. The aspirator is now attached to the needle and the pleural contents withdrawn. To avoid shock this must be done slowly. Finally the puncture wound is sealed with collodion.

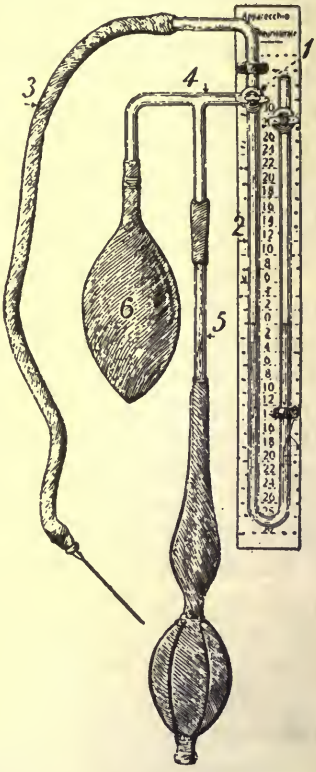

FIG. 855.—MORELLI APPARATUS.

The apparatus consists of a three way cock *1*, one branch of which communicates with a water manometer *2*, one with the pleural cavity *3*, and the third with a Richardson bulb *6*, and a filter *5* (Morelli).

Artificial Pneumothorax.— For the production of *artificial pneumothorax*, Murphy[1] uses pure nitrogen gas. The patient is postured as for paracentesis. Instead of an aspirating needle, a sterile trocar and cannula are inserted down to the rib. The trocar is withdrawn and the cannula connected with a reservoir of sterile nitrogen gas by means of a rubber tube. The cannula is then pushed against the upper margin of the rib into the pleural cavity. The latter is recognized by the flow of gas and the subsequent

compression of the lung. From 500 to 2000 c.cm. of gas may be introduced. The pneumothorax thus produced will last from ten to twenty weeks. The procedure is harmless and may be repeated as the indications arise.

Bastianelli[2] uses the apparatus devised by Morelli. He says "We prefer the apparatus devised by Morelli as being easily transported, and making impossible embolism through technical faults. A glance

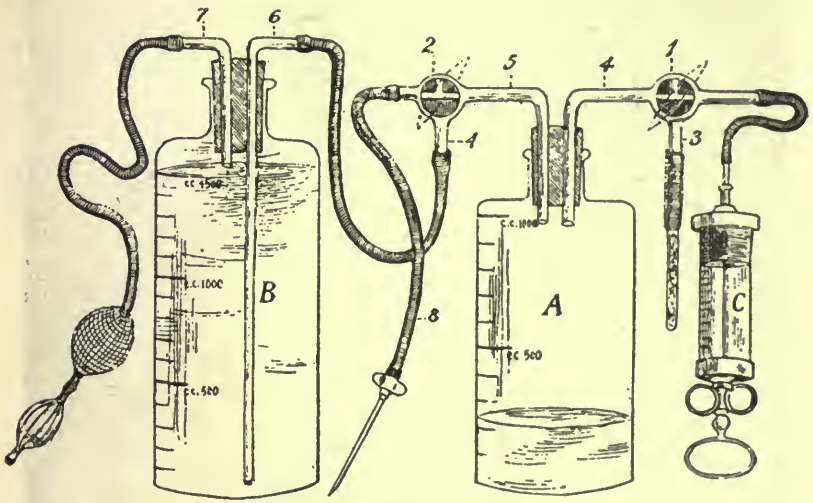

FIG. 856.— MORELLI APPARATUS.

The apparatus consists of a bottle *A* of about one liter capacity. The bottle is closed by means of a rubber stopper through which pass two glass tubes. One of these tubes marked *4* communicates with a three way cock, two branches of which communicate respectively with a syringe *C* of 100 cubic centimeters capacity, and a filter *3*. The second glass tube out of bottle *A* forms one branch of a three way cock marked *2*. One of the other branches of the cock is attached by means of a rubber tube to a thoracentesis needle *8*, and the other communicates with the bottle *B* containing the lavage fluid. Bottle *B* is also closed by means of a rubber stopper through which are two glass tubes, one of which, *6*, communicates with the three way cock *2*, the other, the shorter tube *7*, communicates with a double Richardson bulb (Morelli).

at Fig. 855 will explain its use. The main feature is that the air cannot be blown into the pleural cavity, but is automatically aspirated by the chest from the bag, which has been previously filled with air. The pleural cavity will, according to the pressure existing in itself, aspirate or be expelled into this bag, as if it were the surrounding atmosphere, and in this way there is no possibility of air entering the lung. When blood is to be removed, Morelli uses the apparatus shown

in Fig. 856, which lets out the blood, puts air into the pleural cavity and washes the cavity if desired, and thus makes certain the removal of all blood from the chest."

Artificial pneumothorax is indicated for the relief of tuberculosis,

FIG. 857.— THORACOTOMY: RESECTION OF A RIB.

of chronic pulmonary abscess which has ruptured into a bronchus (provided no pleural adhesions are present to prevent collapse of the lung), in certain cases of bronchiectasis, and in traumatic hemorrhage of the lung.

Thoracotomy.— For thoracotomy, the patient lies on the healthy side

upon a sand bag, which arches the thorax upward and widens the intercostal spaces. As a rule, local anesthesia will suffice. The site of the incision, unless there is some special contra-indication, is the same as that in paracentesis. The incision is made from 4 to 5 cm. in length between and parallel to the ribs, all structures being divided down to the intercostal aponeurosis. The hemorrhage is controlled, and the pleura opened by a small incision and enlarged with an artery clamp or blunt scissors. The fluid is removed; a rubber drainage tube is inserted and held in place with a silk worm gut suture. The method of operation is inadequate if prolonged drainage is required, as the wound usually heals by approximation of the ribs.

Major intercostal thoracotomy without rib resection (Lilienthal[3]) is a comparatively recent procedure. A long intercostal incision is made, usually in the seventh interspace, the ribs being separated from three to five inches or more by means of a powerful "spreader" (Fig. 851). Adequate exposure and ease of manipulation are thus assured, and resection of a rib is rarely necessary. The lung may be collapsed or distended by the anesthetizer who forces either gas or air into the lungs by means of a foot bellows through a tube in one nostril, at the same time closing the patient's mouth and the other nostril.

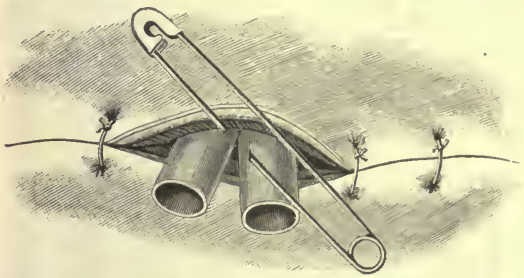

FIG. 858.— THORACOTOMY: DRAINAGE TUBES IN POSITION.

The *indications* for simple thoracotomy are removal of infected effusion in cases where prolonged drainage is not necessary; those for major simple thoracotomy are thoracic exploration and the treatment of lung conditions, etc.

In *thoracotomy with resection of a rib*, the position of the patient, the site of operation, and the incision are the same as in simple thoracotomy. The operation may be performed under local anesthesia. The writer uses narcosis in all but special cases. The incision is from 4 to 5 cm. in length and is made over the rib to be resected. The soft structures are divided down to the periosteum, which in turn is incised and stripped up with an elevator, care being taken not to injure the intercostal vessels and nerve at the inferior border of the rib. Three

to four centimeters of bone are now resected (Fig. 857) with the costotome. After bleeding has been controlled, an incision is made through the periosteal bed of the resected rib, through the intercostal aponeurosis, and through the parietal layer of pleura. The fluid is evacuated gradually, under the control of a large wet pad. Rubber drainage tubes are inserted and held in place as shown in figure 858. The rest of the wound is closed with through and through

FIG. 859.— OSTEOPLASTIC THORACOTOMY.

sutures. If the necessity for further exploration arises, another rib may be resected through the same incision. When the condition of the parietal pleura permits, it may be sutured to the skin (*thoracostomy*) and the drainage tube may be omitted.

When the lung does not expand after more or less persistent drainage of the pleural sac, the following methods may be used:

Osteoplastic thoracotomy for the purpose of exposing the various

sectors of the thoracic cavity, as applied to the anterior region, may be described as follows: A vertical incision, from 12 to 15 em. in length is made parallel to, and from 1 to 2 em. lateral to, the border of the sternum through the soft parts to the costal cartilage; two horizontal incisions are made extending outward over the ribs from the two poles of the vertieal incision through the soft parts down to the costal cartilages and ribs. The internal mammary artery is ligated above and below the costal cartilages; the latter are seetioned near the sternum; the remaining soft parts and parietal pleura are divided along the line of the incisions, and the rectangular flap thus made is raised and bent outward, fracturing the ribs at the external aspect of the flap (Fig. 859). After the intrathoracie manipulations are completed. the flap is sutured in plaee.

FIG. 860.— GREEN'S METHOD OF PNEUMECTOMY.

The portion X is cut away, leaving a V-shaped wound of lung, which is sutured, as shown in figure 861, bringing the edges of the wound together and coapting the pleural edges, as in figure 862 (but not apposing the serous surfaces, as in the Lembert suture of the peritoneum). This covers in the ligating suture and shuts off the divided bronchial tube (from Keen's Surgery, copyright by W. B. Saunders Co.).

Decortication of the Lung.— Decortieation of the lung (Fowler[4]) is praeticed for the purpose of occluding persistent empyemie cavities. The pleural eavity is exposed by reseetion of several ribs, or by an osteoplastie flap, or by major thoraeotomy, with or without rib resection. An ineision is made through the fibrinous exudate on the surface of the lung and this is stripped away with seissors. In favorable eases, the lung expands immediately. The best results are obtained in ehildren and in cases that are attaeked in this way soon after primary

drainage and before cirrhosis of lung tissue occurs. In certain instances, it is impossible to strip the membrane without injury to lung tissue.

Thoracoplasty.— Thoracoplasty (Estländer[5]) consists in the removal of several ribs immediately over a residual empyema cavity, with the view of approximating the wall of the thorax to the lung. The ribs are exposed by a double rectangular flap. A U-shaped incision or several horizontal incisions are made over the intercostal spaces, through each of which two ribs may be resected. The amount of rib removed depends upon the size of the cavity. Recently Estländer advocates removal of the intercostal muscles, the aponeurosis, and thickened parietal pleura, and advises scraping the pyogenic membrane from the lung surface.

FIG. 861.— GREEN'S METHOD OF PNEUMECTOMY.
Ligating suture tied; distal portion of lung cut away; the coapting suture introduced ready to be drawn tight (from Keen's Surgery, copyright by W. B. Saunders Co.).

Schede[6] removes from the second to the tenth or eleventh rib, together with the intercostal muscles, fascia and parietal pleura. The incision, which is made from the costal cartilage of the second rib, downward along the cartilages to the tenth rib, is then carried outward along the upper border of the latter to a point near its angle and then upward along the vertebral border of the scapula to the second intercostal space behind. The superficial structures are divided down to the ribs and the flap, including the scapula and the attached muscles, dissected up. After the bleeding has been controlled, the costal cartilages are divided in front, and then the ribs are sectioned near their tubercles. The

intercostal arteries are ligated, and finally the ribs, intercostal structures, and parietal pleura are cut away with heavy scissors. The visceral pleura is curetted and the skin flap, together with the superficial layers of the soft parts, is apposed to the contracted lung. The flap is held in place by two layers of sutures, one in the musculature, the second in the skin. The dressing should be applied in such a way as to maintain contact between the contracted lung and the flap.

Pneumectomy.— Pneumectomy follows exposure of the diseased or injured lung by one of the above methods. The lung is delivered through the opening made in the chest wall and held there with one or two traction loops placed behind the doomed sector of lung tissue, or the latter may be encircled with a heavy silk suture introduced with a blunt, round, curved needle (Green[7]).

Anterior Mediastinal Thoracotomy.— Anterior mediastinal thoracotomy is, at the present time, performed by splitting the sternum. A cutaneous incision is made in the midline from a point just below the thyroid cartilage extending down to the ensiform. The upper part of the incision is gradually deepened until the trachea is exposed; its lower portion is carried down to the sternum, and the gladiolus is separated from the ensiform. The soft parts are separated from the posterior surface of the sternum with the finger or blunt periosteal elevator, hugging the bone and being careful not to injure the pleura or great vessels, especially the veins. The sternum is sectioned in the line of the superficial incision by means of a saw, the underlying structures being protected by a steel spatula. In the central portion of the sternum it is necessary only partially to divide the bone; its posterior surface may be cut with bone forceps or fractured as the edges of the sawed surface are drawn apart. The latter are now gradually separated to the extent of from 6 to 8 cm., which is made feasible by carefully pushing aside the underlying structures. This

FIG. 862.— GREEN'S METHOD OF PNEUMECTOMY.

Operation completed (from Keen's Surgery, copyright by W. B. Saunders Co.).

gives ample space for the necessary intramediastinal manipulations. When these are completed, the divided bone is approximated by means of three or four chromic gut sutures passed through drill holes. Following this the superficial wound is sutured.

Posterior Mediastinal Thoracotomy.— In *posterior mediastinal thoracotomy* (Bryant[8]) the patient is placed in the Sims position, the operative side uppermost and the arm raised well over the head to widen the intercostal spaces. As in the osteoplastic methods, the site of the operation depends on the location of the lesion. A square is simulated, the base of the flap, which is 10 cm. in length, corresponds

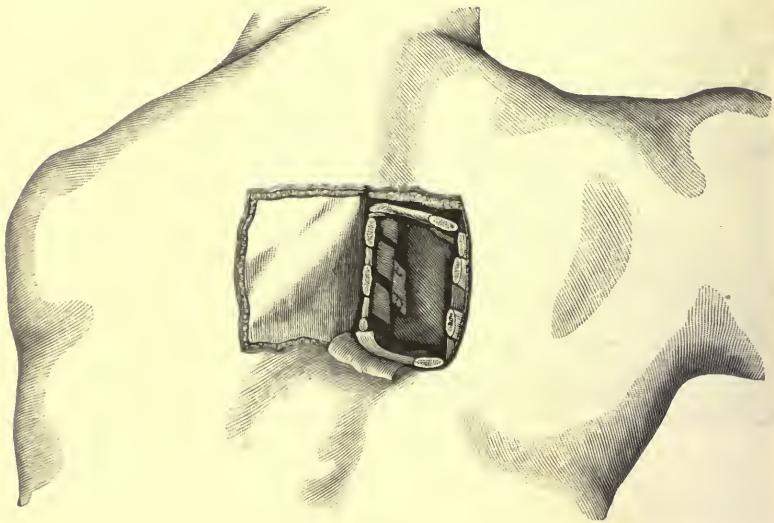

FIG. 863.— POSTERIOR MEDIASTINOTOMY (Bryant).

to the line over the spinous processes; two horizontal incisions, 10 cm. long, are made, extending laterally, and are connected at their outer extremities by a vertical incision which measures 10 cm. All the incisions are carried to the bone, the flap is raised and retracted, carrying the underlying muscle with it, thus exposing three ribs from their angles to the junction with the transverse processes of the vertebrae. The middle rib is resected subperiosteally, and the upper and lower ribs are divided at each end without removing either muscles or fascia. A horizontal incision is now made through the periosteum of the resected rib and the pleura is separated from behind the upper and lower ribs and intercostal muscles with the finger. Two lateral in-

cisions are made at right angles to the horizontal, making an H-shaped incision separating the intercostal muscles and fascia. The two flaps thus formed are now raised and retracted upward and downward respectively. The pleura is further separated from the bodies of the vertebrae, and the mediastinal structures are exposed. After the necessary mediastinal procedures are completed, the rib flaps are sutured in place and the musculocutaneous tissues closed. Drainage should be established, if the œsophagus or a bronchus has been opened.

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CHAPTER VII

THE DIAPHRAGM

Congenital Malformations.— Of the congenital malformations of the diaphragm, the *defects are of surgical importance.* The defect may involve all the layers of the diaphragm, or one layer or more may be absent. In the former, a free communication exists between the thorax and the abdomen and the contents of the latter protrude directly into the thorax, or the protrusion may be covered with the pleura and the peritoneum alone. The condition is classified as a *congenital diaphragmatic hernia.* A second class of hernia (relaxatio diaphragmatica) is described by Eppinger.[1] It consists in the bulging of the practically intact diaphragm, together with more or less of the abdominal contents, into the thoracic cavity, without any solution of continuity of the diaphragm. The condition is probably congenital in origin.

Diverticulum of the diaphragm is a term applied to restricted sacculated areas, which are usually located in the tendinous portion of the muscle and at times give rise to the clinical picture of hernia.

Injuries of the Diaphragm.— Punctured wounds of the diaphragm, the outcome of stab or gunshot wounds, are very common. The muscle alone is rarely injured. As a rule the contents of the abdomen or the lung are also involved.

The injury is not attended with pathognomonic *symptoms.* The clinical picture of the injury to the other parts usually dominates the situation. However, prolapse of the omentum through a wound in the thorax makes the diagnosis certain. The *diagnosis* is also patent when there is evidence of simultaneous involvement of the thoracic and abdominal contents in cases of gunshot or stab wounds. For instance, the signs of a pneumothorax or of abdominal hemorrhage leave no doubt in this connection.

The *prognosis* in this class of cases is usually determined by the severity of the coexisting injuries. However, the part that the diaphragmatic injury by itself plays in this connection depends upon whether the muscle fibers are merely separated or are divided trans-

versely. In the latter instance, respiratory movements tend to increase the wound area, and hernia is very likely to occur.

The *treatment* of perforating wounds of the diaphragm, in which the diagnosis is made or is suspected, consists in operative repair of the lesion. This is best accomplished by *thoracotomy* (p. 1849), which makes the entire surface of the diaphragm readily accessible and also permits of repair of the injury to the abdominal organs (*transpleural celiotomy*) by enlarging the wound in the diaphragm (Schumacher[2]). On the other hand, *celiotomy* is indicated when the clinical evidence of injury to the abdominal contents dominates the picture.

Hernia of the Diaphragm.— The term *hernia diaphragmatica vera* is applied to protrusions of the abdominal contents into the thorax covered by peritoneum or pleura. When neither of these coverings is present, the term *hernia diaphragmatica spuria* is used. In the latter instance the abdominal invaders lie free in the thoracic cavity. Schumacher[2] makes the following division of the subject:

1. *Herniae diaphragmaticae verae.*
   - i. *Herniae diaphragmaticae verae congenitae,* which are characterized by a protrusion through a congenital defect.
   - ii. *Herniae diaphragmaticae verae acquisitae,* which protrude through normal openings in the diaphragm, i. e., esophageal, aortic, etc.
     - a. *Herniae diaphragmaticae verae sternalis,* which protrudes between the pars sternalis, pars costalis and the sternum or seventh rib (foramen Morgagni).
     - b. *Herniae diaphragmaticae verae para-esophageae.*
     - c. *Herniae diaphragmaticae verae foraminus Bochdalecki,* which protrude through a triangular opening in the diaphragm bounded by the last rib and the posterior costal origin.
   - iii. *Herniae diaphragmaticae verae atypicae.*
     - a. Those protruding through an apparently normal diaphragm.
     - b. Those protruding through a diaphragm which has been the site of pathological processes of various kinds.
     - c. *Herniae diaphragmaticae verae traumaticae,* in which the protrusion escapes through a solution of continuity of the diaghragm caused by an injury.

2. *Herniae diaphragmaticae spuriae.*
   i. *Herniae diaphragmaticae spuriae congenitae* consist in congenital absence of the diaphragm. As children afflicted in this way do not survive, the condition possesses no surgical importance.
   ii. *Herniae diaphragmaticae spuriae traumaticae,* already alluded to in connection with injuries, may occur immediately after receipt of the trauma or develop later as the result of the consequent weakening of the diaphragmatic musculature.

The *diagnosis* of the presence of abdominal contents in the thorax may be made when a recent injury of the latter is followed by the clinical picture of intestinal obstruction. The only time when the diagnosis is made in the other forms of hernia is when the symptoms of intestinal obstruction are presented and exploratory exposure of the lesion is made. On the other hand, if the condition is recognized by the history of the case, as is not unlikely when abdominal symptoms follow closely upon a thoracic injury, the surgical attack should be made by means of a *thoracotomy.* Schumacher[2] advises that, if celiotomy reveals the presence of a diaphragmatic hernia, reposition of the protrusion and repair of the diaphragm be effected through an additional thoracotomy opening made at the same time. See also Salomini[3] and Gross[4].

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CHAPTER VIII

SURGERY OF THE PERICARDIUM AND HEART

THE PERICARDIUM

Puncture of the pericardium, as practiced by Dieulafoy (1869) who trephined the sternum, was abandoned, to be revived by Porter,[1] Paget,[2] and others.

Paracentesis of the Pericardium.— *Paracentesis pericardii* is practiced for diagnostic purposes and for evacuation of serous or purulent exudates. When the pericardial sac becomes filled, the heart and lungs are displaced; the heart is raised and its apex rotated forward and the precordial area is enlarged. Dieulafoy[3] placed his patients in a semirecumbent position and selected a point about 6 cm. from the left border of the sternum in the fifth interspace. No. i or ii needles were used, preferably the latter. After penetrating 1 cm., aspiration is started by opening the stopcock which leads to the vacuum already established in the syringe (Fig. 364). The needle is gradually advanced from above downward until fluid appears in the glass index of the aspirator. As the flow diminishes, the needle is tilted horizontally so as to make it run parallel to the ventricle and avoid cardiac contact.

The diagram shown in Fig. 864 indicates the several sites of puncture utilized by various surgeons. The mammary vessels and the pleura are avoided. The procedure is unreliable in all cases of doubtful diagnosis. In the latter instance, pericardiotomy is preferable.

Pericardiotomy.— Pericardiotomy is practiced in cases of doubtful diagnosis, but especially for the relief of purulent pericarditis. A number of methods, not all of which can be related here, are used for the purpose. Matas[4] says "After considering the various methods consistently with reasons which lead me to select the immediate left lower sternal and parasternal region as the safest and best for paracentesis, I must again express my preference for this route for pericardiotomy, just as we must aim, in puncturing the pericardium, to

select that spot which will avoid the blood vessels and the pleura and yet strike the pericardial accumulation where it is largest and most dependent and where, in addition, the lower sternophrenic reflection of the pericardium will insure the greatest protection to the heart."

Narcosis is indispensable in young and nervous persons; in others, local anesthesia may be used. The following procedure is known as the Allingham operation, modified by Pendleburg:[5] A vertical incision 4½ in. long is made through the skin and fascia; the center of the incision is located at the lower border of the seventh costal cartilage, parallel with, and 1 in. from, the left border of the sternum. The skin and fascia are reflected and the rectus and oblique abdominal muscle are detached from the seventh costal cartilage. The cellular space thus exposed is broken into with the finger and some of the fibers of the diaphragm are torn. This costoxiphoid space, containing the superior epigastric artery, is enlarged by cutting away a part of the left seventh costal cartilage with a rongeur, and if further room is required, the sixth cartilage is also resected. The internal mammary vessels are now exposed and ligated, if necessary, and the triangularis sterni with the connective tissue and fat and the right and left pleura, is carefully pushed aside. To avoid entering the peritoneal cavity, the pericardial sac should be entered at the level of the base of the ensiform cartilage by a blunt instrument, and the opening enlarged downward into the costoxiphoid space with the finger. A soft rubber drainage tube is inserted and left *in situ* from twenty-four to thirty-six hours.

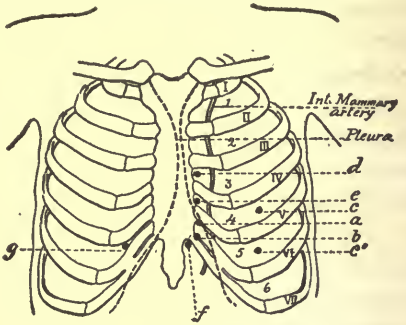

FIG. 864.— POINTS OF ELECTION IN PERICARDICENTESIS.

The dotted line shows pleura. *a*, Baizeau; *b*, Delorme and Mignon, Hare, Voinitch-Sianojentsky; *c* and *c*¹, Dienlafoy; *d*, Schuh; *e*, Trousseau; *f*, Larry; *g*, Rotch, Wilson, etc. (from Keen's "Surgery," copyright by W. B. Saunders Co.).

Brentano[6] and Porter[1] recommend subperiosteal resection of the fifth left costal cartilage. Durano[7] modifies this by resection of the sternum, if a greater exposure is desired. Delorme and Mignon[8] resect the fifth and sixth left costal cartilage by a vertical incision, while

v. Eiselsberg[9] resects the fourth left cartilage. Bacon,[10] of Cleveland, recommends trephining the sternum.

Cardiolysis.— Cardiolysis is practiced in certain forms of adhesive or plastic pericarditis. Brauer[11] recommends the removal of the overlying osteocartilaginous wall in front of the pericardium, which changes the rigid precordial region into a soft, yielding covering. Peterson and Simon[12] performed this operation with marked improvement in three cases. Peterson recommends removal of three left costal cartilages with small portions of their ribs and part of the sternum.

Wounds of the Pericardium.— Wounds of the pericardium usually correspond to the size of the superficial skin wound. In crushing wounds with fracture of the ribs and the sternum, the rent in the pericardium is likely to be large, although a large tear of the pericardium may occur without fracture of these bones. The *treatment* is described under Wounds of the Heart.

Foreign Bodies in the Pericardium.— Foreign bodies in the pericardium are likely to be projectiles and needles, the latter entering from the esophagus. The projectiles are found free or encysted in the cavity. Suppurative pericarditis follows if the foreign body is contaminated.

THE HEART

Wounds of the Heart.— Wounds of the heart are divided into *non-penetrating,* occurring in connection with contusions and crushing injuries which lead to laceration and rupture; and *penetrating,* produced by projectiles or cutting instruments. The latter may involve one or more of the walls of the heart. To these may be added pathological rupture of the heart caused by degeneration of its muscle fibers. Penetrating wounds make up 25 per cent of the cases. Fischer[13] classifies wounds of the heart as he does general wounds, i. e., contused, lacerated, incised, etc., any of which may be penetrating or perforating.

The right ventricle is most often involved; then the left ventricle, the right auricle, and the left auricle, in the order mentioned. According to Loison,[14] gunshot wounds involve the left ventricle more often than the right. Wounds of the auricles are more likely to be fatal immediately than those of the ventricles.

Of the *symptoms,* Matas[4] says "While there is no pathognomonic sign by which a wound of the heart can be recognized, there are, however, groups of signs and symptoms which, when combined with the history of the case, the seat and direction of the wound, or the prob-

able range and track of the missile (if it is a gunshot wound) make the diagnosis reasonably certain."

If a fatal outcome does not immediately occur from cessation of the heart action or intrapericardial pressure from effusion of blood or from external and internal hemorrhage, the patient presents the picture of profound shock attended with distressing dyspnea and severe pain. As in all injuries of the chest, the gravity of the symptoms is not proportionate to the extent of the wound. A comparatively slight injury may be accompanied at times by the most formidable symptoms, even when there is no cardiac involvement. On the other hand, the apearance of the patient may not be menacing, although the actual cardiac lesion is exceedingly grave. As a rule, however, the symptoms present the classic picture of external or internal hemorrhage, the former expelling an intermittent stream of blood with each heart beat, the latter expressed by hemothorax. Both produce acute anemia, progressive dyspnea, and great prostration. A small pericardial opening may be spontaneously occluded so that hemorrhagic effusion into the pericardium ensues. In this event, the heart sounds become less and less perceptible as the bloody effusion increases, while the pressure upon the auricles causes engorgement of the general venous system. Occasionally, bulging of the anterior thoracic wall may be noted. This syndrome is called "heart tamponade" (Rose[15]).

The *diagnosis* is not difficult when the provocative weapon protrudes from the chest wall and moves synchronously with the heart beats; nor when a large gaping wound exposes the heart and a stream of blood pours out of the opening. However, in cases in which there is the disproportion of symptoms stated above, the diagnosis must rest on the history, the location of the superficial wound (in cases of gunshot wound, on the direction of the projectile), the evidences of bleeding or heart tamponade, together with the physical signs.

With respect to *prognosis,* the cases may be divided into two classes, the first, in which the signs and symptoms indicate the rationale of operative measures of relief; and the second, in which the absence of menacing symptoms justifies a policy of non-interference, with the indefinite possibility that spontaneous healing may occur. However, in the latter class of cases a fatal outcome often results from sudden secondary hemorrhage or from infection of the pericardial sac.

The *treatment,* in cases in which non-interference is decided upon, consists in absolute rest and the administration of opiates.

As in pericardiotomy, the methods of *operative repair* are numer-

ous. Simple intercostal incision and spreading of the ribs, as described under thoracotomy, has been practiced by Spongaro.[16] Thoracotomy with resection of ribs and the formation of osteoplastic flaps, which includes median section of the sternum (Fontan[17]), have also been employed. Of the various methods — some of which have already been discussed in connection with pericardiotomy — that of Kocher[18] is perhaps most satisfactory in cases in which the diagnosis is not clear. An angular incision is made, its perpendicular limb corresponding to the midsternal line; the transverse limb extending beyond the left sixth costal cartilage. The periosteum of the sternum and perichondrium of the cartilage, together with the attachment of the pectoralis major muscle are pushed aside and the cartilage is freed on its posterior surface. The cartilage is now severed near the sternum; the soft parts in the area of the transverse incision are separated still farther from it and the cartilage is resected. The attachment of the triangularis sterni is next separated from the sternum and the internal mammary vessels ligated. The pericardium may now be felt through the opening in the sixth costal cartilage and punctured or incised. By pushing aside the intercostal fascia with the internal mammary vessels and pleura, the heart may be explored with the finger. If the injury to the latter is thus located, the fifth, fourth, and third costal cartilages are divided at their costal edges and turned outward with the triangular flap. Additional space may be obtained by carrying the transverse incision farther outward, fracturing the ribs at the mammary line and turning the flap thus formed outward. When the diagnosis is not in doubt, the simplicity of the method practiced by Spongaro[16] recommends its employment. The incision is made in the center of the fourth intercostal space, its median end beginning near the sternum and its distal terminating at the anterior axillary line; the mammary vessels are ligated at once. The ribs are readily forced apart, as described in connection with thoracotomy (p. 1849), exposing the pericardium, the pleura and the lung. Additional space may be obtained by dividing the sternal ends of the costal cartilages above and below the intercostal incision.

Luckett (personal communication) found no difficulty in removing a bullet from the heart by intercostal thoracotomy, employing a technic (Fig. 865) not unlike that of Spongaro. Although the immediate postoperative period was stormy, ultimate recovery was complete.

If the pericardial sac has been opened by the original injury, heart tamponade is not present and repair of the injury to the heart may be

proceeded with. When the pericardium is filled with blood, it is rapidly opened from the apex to the base of the heart and the contents are evacuated. The edges of the wound in the sac are grasped with forceps and with slight traction the heart is easily brought into view and its repair made feasible.

When the heart action is violent, the operative field is likely to be flooded with blood which, when the pleura is also injured, is churned

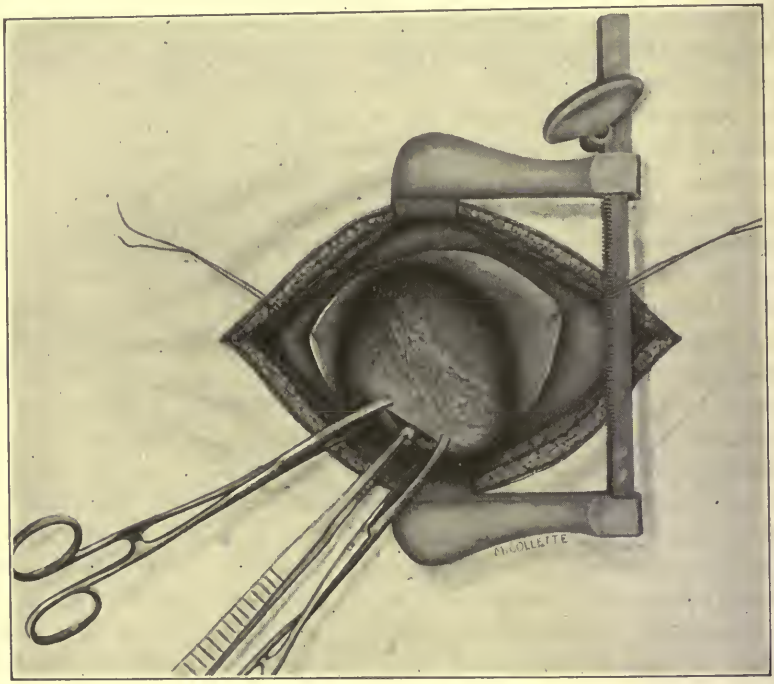

FIG. 865.— INTERCOSTAL PERICARDIOTOMY: EXTRACTION OF BULLET FROM RIGHT VENTRICLE (Luckett).

into froth at each respiration. In these instances the hand is immediately introduced into the pericardial sac, the wound located and promtply occluded with the finger. If this is not feasible, the heart is delivered and alternately compressed and released until the site of the wound is made evident (Matas[19]). While the wound in the heart is being repaired, it is best steadied with the hand; instumental fixation causes additional trauma. The repair is best made by means of interupted chromic gut sutures introduced with a small, curved in-

testinal needle. Drainage of the pericardial sac is of great importance; infection is always fatal (Guibal,[20] Tscherniachowski[21]).

Foreign Bodies in the Heart.— Foreign bodies in the heart, if their entrance is not immediately fatal, are likely to be well borne, unless infection supervenes. To prevent infection, they should be removed. The technic of the procedure is somewhat similar to that discussed in connection with wounds of the heart.

A wound of a single coronary artery is not necessarily fatal (Porter[22]). Capelen[23] tied the left anterior ascending coronary artery in the living human. The patient died on the fifth day from sepsis.

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CHAPTER IX

THE SURGERY OF THE BREAST

Malformations of the Breast.— Congenital anomalies of the mammary glands are not uncommon and are ascribable to disturbances in development. They occur in both sexes and are not infrequently associated with malformations of other portions of the body.

Amastia, i. e., total absence of mammary gland tissue, is very rare, while *polymastia* — the presence of more than two glands — is less *uncommon.* The adventitious gland is usually situated either above or below the normal one, but may be located in the axilla, the thigh, of the buttocks. As a rule, it is rudimentary in character, but occasionally secretes milk. The nursing of an infant from the buttock has its disadvantages; this and other cosmetic reasons justify extirpation of the adventitious gland.

Gynecomastia, enlargement of the male breast, may be single or double (Fig. 528). Genuine gynecomastia consists of an increase of stroma and parenchyma. The folds of fat seen in some instances are not the same thing. *Extirpation* is indicated for cosmetic reasons.

Athelia is a term applied to partial or complete absence of the nipple. In some instances the nipple is bifid. *Polythelia* refers to the presence of more than one nipple attached to the same gland.

Diseases of the Breast.—Hypertrophy of the mammary gland consists in a diffuse painless thickening of the fibrous stroma and parenchyma. At times, the breasts become enormous (Fig. 866), but lactation is diminished or absent. Both or only one breast may be involved. The etiology of the process is not understood. *Amputation* of the breast is indicated.

Inversion of the nipple may be congenital, or may be due to the contraction of the fibrous tissue of the gland which occurs in connection with inflammatory lesions or with the development of a neoplasm, especially carcinoma.

Fissures of the nipple are due to uncleanliness during the last months of pregnancy. Infection of a fissure is attended with the

formation of abscess. The primary condition demands frequent cleansing with boric acid solution and the occasional application of silver. Abscesses are drained.

Eczema of the nipple is important because of its frequent association with malignant disease of the breast (Paget's disease).

Aveolar abscesses usually develop in girls at puberty. They originate in the sebaceous follicles and should be promptly opened.

Tumors of the nipple are not frequent. They are usually sebaceous

FIG. 866.—HYPERTROPHY OF THE BREASTS (Symmers, Bellevue Hospital case).

cysts, papillomata or angiomata. Epitheliomata occasionally develop in this situation.

Chancre of the nipple is rarely seen in the nursing mother of a luetic child, but appears upon the wet nurse.

Of the *wounds of the breast,* the incised and lacerated are likely to be followed by the formation of a milk fistula, while contusions are often attended with the development of large hematomata, which very often become infected and suppurate.

In milk fistula, lactation should be arrested, the tract excised, and careful repair made by suture. Hematomata are best incised early.

Mastitis neonatorum and mastitis adolescentium are really physiological conditions attended with mild hypertrophy of the gland tissue accompanied by the phenomena of inflammation. As a rule, the conditions subside promptly; occasionally, however, they go on to suppuration and must then be treated as such.

Stagnation mastitis (caked breast) is the outcome of retention of the lacteal secretion. It occurs late in pregnancy or at the end of lactation. Usually the process in manifested in a single lobe which becomes indurated and exhibits the phenomena of inflammation.

Suppuration may often be prevented by emptying the breast with the suction pump, or by massage and the maintenance of firm pressure. When abscess formation occurs, free incision is indicated. This must not be delayed.

Acute mastitis, the outcome of infection, occurs most frequently in the nursing mother, though it also develops in the nonlactating breast by extension from the nipple. It may also be hematogenous in origin.

It is differentiated from retention mastitis by the primary appearance of the evidences of inflammation. The process may be entirely interstitial or intra-areolar or present a combination of the two. Low grade infections may end in spontaneous regression of the process. However, as a rule it goes on to suppuration, and at times the entire gland is destroyed. Pus collects in one of three places: (a) in the subcutaneous tissue just below the nipple, with or without involvement of the breast proper; (b) in the lobules and in the perilobular tissues, in which event the inflammation is diffused throughout the entire gland, forming multiple abscesses and causing more or less destruction of the gland structures; (c) in the cellular tissue behind the gland where, as a rule, it is secondary to caries of the ribs or to a deep seated cellulitis. It may also be secondary to a deep lobular infection of the breast proper. In the latter event, the breast is pushed forward and the pus points at the periphery of the base of the breasts.

The *treatment* during the early stages of the process consists in immobilization of the breast and the application of an ice bag. Upon the first evidence of suppuration, the breast must be freely incised. Delay in this connection is not permissible. See Inflammation, Part II, chap. ix. The incision should radiate from the nipple and free drainage must be provided for. In the retromammary type of suppuration, a semicircular incision at the lower aspect of the gland accomplishes the purpose and obviates offensive scarring.

Chronic mastitis, in view of the resemblance it bears to malignant disease, is of great surgical importance. It may be classified as follows:

1. Chronic lobar mastitis.
2. Chronic lobular or interstitial mastitis; chronic cystic mastitis (Koenig[1]); cystadenoma of the breast (Schimmelbusch[2]); cystic disease of the breast (Reclus[3]).
3. Tuberculous mastitis.
4. Luetic mastitis.
5. Actinomycosis.

Chronic lobar mastitis is characterized by an enlargement of one or more lobes of the breast, attended with pain and tenderness. The condition develops at the end of lactation and usually disappears spontaneously.

Chronic lobular, or cystic mastitis (see also cystadenoma), is veiled in a cloud of mystery. Koenig[1] regarded the condition as inflammatory in character. Recently the view that it is a parenchymatous hypertrophy, the result of abnormal involution, has gained ground. The importance of the process lies in its tendency to assume the characteristics of malignant disease. This is especially true as the affection develops in the "cancer age." Finney[4] presents an exhaustive study of the pathology of the condition. He regards the presence of smooth walled cysts as distinctive. The cysts are usually multiple and vary widely in size. The walls are at first lined with a layer of columnar epithelium, which later disappears. The surrounding parenchyma and periglandular connective tissue present a condition of low grade inflammation.

In a certain number of the cases the glandular acini and the cysts are filled with papillary outgrowths simulating adenomata.

As the fibro-adenomatous changes progress, the process takes on an infiltrating form WHICH ULTIMATELY CANNOT BE DIFFERENTIATED FROM ADENOCARCINOMA.

The *clinical picture of lobular mastitis* is not distinctive. The early stages are not attended with symptoms. The presence of a mass usually attracts attention. The breast soon enlarges and becomes painful. The nipple, as may be imagined, is often retracted and discharges a serous fluid. A mass may be present in both breasts; the skin is rarely adherent and occasionally small masses are present. The axillary lymph nodes may be enlarged and tender, but are not hard. Finally, the mass is not well defined, but imparts to the palpating fingers the sensation of its merging into the surrounding tissues. It

does not present the hardness of scirrhus nor is it attached to the pectoral fascia.

The *treatment* is based on the tendency the process possesses to become malignant, and consists in amputation of the breast or breasts.

Chronic encysted abscess of the breast is characterized by the presence of an indurated mass which later undergoes softening. The thickened walls of the process may cause it to be mistaken for a tumor. Aspiration clears the diagnosis.

Incision and curettage is followed by healing.

Tuberculous mastitis occurs in the form of an encysted cold abscess — as described above — or in the form of caseous nodules which break down and form sinuses. Occasionally, tuberculous osteomyelitis of a rib extends to the breast.

The *treatment* consists in free incision and curettage of the infected areas, or, if a considerable portion of the breast is involved, amputation of the gland is indicated.

Luetic lesions of the breast, other than chancre of the nipple, are rare, and appear in the form of gummata in the tertiary stage of the disease. The lesion must be differentiated from tuberculosis and other chronic inflammatory processes.

Actinomycosis of the breast is rare. It bears much resemblance to tuberculosis and can be differentiated from it only with the aid of the microscope (Part II, chap. xix).

Benign Tumors of the Breast.— All of the benign tumors found elsewhere in the body make their appearance also in the breast. Only those peculiar to this situation are considered here.

Adenofibroma, excluding carcinoma, is the most common tumor of the breast. As a rule, the fibrous elements dominate, although the glandular structure occasionally predominates. Adenofibromata occur as a more or less round or oval mass (or masses), which is freely movable, does not cause pain, and is not attended with enlargement of the axillary lymph nodes, nor is the nipple retracted. Extirpation is easily accomplished.

Cysto-adenoma bears considerable resemblance to chronic lobular mastitis. It is characterized by cystic dilatation of the acini of the breast, the interiors of which are lined with masses of newly formed adenomatous tissue and the external surfaces surrounded by proliferated fibrous tissue. As a rule, several cysts are present, inclosed in a definite capsule. A single cyst usually contains pedunculated masses of proliferated epithelium. Cysts of this sort are very vascular

and when hemorrhage occurs as it frequently does, a bloody fluid is likely to be discharged from the nipple.

The tumor is irregular or lobulated, painless, and rarely attended with enlargement of the axillary glands. As the tumor enlarges, it is likely to perforate the skin, ulcerate, and present a fungous mass which bleeds readily.

The *treatment* consists in extirpation of the tumor, unless it has attained great size, when amputation of the breast is indicated.

Cysts of the breast abound in great variety. It would seem permissible to divide them into those peculiar to the mammary gland and those similar to cysts which appear elsewhere in the body. The former is far more common than the latter.

Type one is divided into: (1) Retention cysts; (2) simple cysts; (3) cysts with papillomatous growths; and (4) cancer cysts, and cysts developing into cancerous growths.

To *type two* belong hydatid and dermoid cysts.

Retention cysts (acinus glactocele) develop in connection with obstruction of the milk ducts and occur during lactation. At first the cysts contain pure milk; later, the fluid constituent of the contents is gradually absorbed, leaving a cheesy residue. The cyst is usually situated behind the nipple, and fluctuates early in the process. There is little, if any, disturbance unless infection supervenes. In the latter event, incision and drainage are followed by healing. In the absence of infection, exposure of the cavity and curettement or, better still, extirpation of the sac is indicated.

Simple cysts occur in idle breasts and, as a rule, develop after the fortieth year of life. The cysts may be single or multiple and often appear in both breasts. They are bluish in color and present a smooth glistening surface. In not a few instances, the process becomes malignant in character, especially when associated with senile parenchymatous hypertrophy.

When the outline of the cyst is smooth and its contents is devoid of blood, extirpation may be practiced. The presence of multiple cysts surrounded by indurated tisssue is an indication for complete excision of the breast.

Cysts with intracystic papillomatous growths present a picture identical with the late stages of the process described in connection with chronic lobular mastitis (Koenig[1]). Malignant changes occur in fifty per cent of the cases. The latter is heralded by infiltration of the breast tissue surrounding the walls of the cysts.

The *clinical picture* and the physical findings are those of simple cysts, except that the nipple often discharges a bloody fluid.

Prompt extirpation of the entire breast is indicated.

Cancer cysts (so-called) are taken up in connection with carcinoma.

Hydatid cysts do not present a distinctive clinical picture. The *diagnosis* is made by examination of aspirated contents or at the operation.

Dermoid cysts are rare and, like hydatid cysts, are not recognized as such until during or after removal.

The *clinical characteristics of benign growths* may be summarized as follows: the benign tumor is encapsulated and freely movable. There is no dimpling of the skin nor retraction of the nipple. Multiplicity of the lesion argues for benignity. Benign growths are usually soft in consistency, occur early in life, and are often attended with pain; the discharge of fluid from the nipple is rarely bloody.

The tendency benign tumors of the breast show to become malignant argues for prompt extirpation. The extent of the surgical measure depends upon the character of the tumor. If it be of a class which often becomes malignant, the radical operation is indicated.

Malignant Tumors of the Breast.— Sarcoma of the breast is rare and is more likely to be *primary* than *secondary*. Primary growths arise from the intralobular connective tissue or from the interstitial tissue of the gland. The spindle cell variety predominates, although round cell and occasionally melanosarcoma develop in this situation. It is probable that the intralobular type, which is the most common, represents a late stage of myxoma.

At first, sarcoma of the breast makes its appearance in the guise of a benign tumor. Later, the tumor undergoes cystic degeneration and invades the entire gland structure and the pectoral fascia. Ultimately, the process breaks down, ulcerates and invades the axillary lymph nodes. At this stage of the disease, metastases develop, especially in the internal organs.

The *distinctive features* of sarcoma of the breast consist in its rapid growth, early cystic degeneration, the absence of retraction of the nipple, and the activity of the process without primary glandular invasion.

Prompt execution of the radical operation is imperatively indicated.

Carcinoma of the breast has already been extensively discussed in

the general part of this work (Part VI). Attention is called to the following *anatomical considerations.* The breast is not a sharply defined organ, but extends along Cooper's ligament to, and into, the pectoralis major muscle and merges with the surrounding fat (Oelsner,[5] Heidenhain[6]). The gland communicates freely with the *lymphatic system.* The lymphatics of the *skin* communicate directly with the axilla and

FIG. 867.— SARCOMA OF THE BREAST (Haynes).

with the supraclavicular region. The lymph channels of the fat are not demonstrable but probably accompany the blood vessels to the axilla and the thoracic cavity. The *lymph vessels of the gland proper* are arranged as follows: those from the periphery enter efferent channels, some of which (the superficial) join the cutaneous lymphatics, others (the deep) extend to the pectoral fascia. Another group,

which drains the lower quadrants of the breast, accompanies the perforating branches of the internal mammary vessels, drains into the lymph nodes of the anterior mediastinum, follows the course of the intercostal vessels, and empties into the glands of the vertebral region. An important lymphatic (not always present) pierces the pectoralis major muscle, extends between it and the minor muscle and enters the anterior mediastinum through the second intercostal space. Another set parallels the fibers of the pectoralis major muscle and enters the mediastinum near the sternum. The lymphatics of the clavicular region drain into the retropectoral glands; those of the manubrial region pass

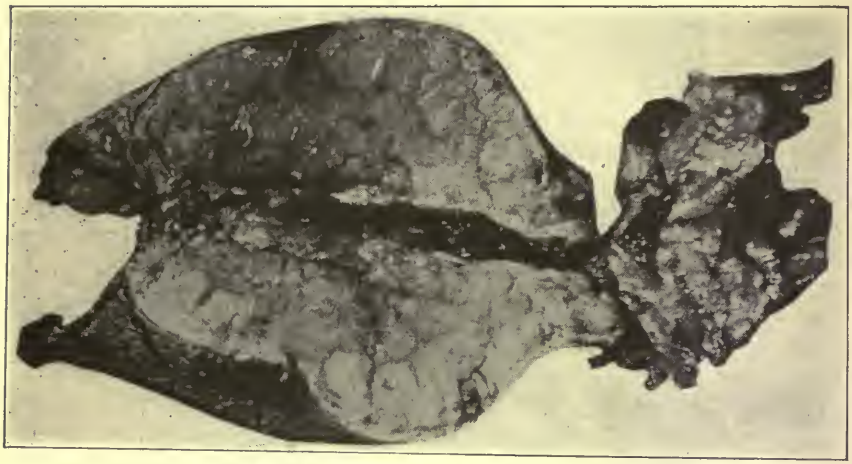

FIG. 868.— SECTIONED CARCINOMA OF THE BREAST, TOGETHER WITH THE AXILLARY LYMPH NODES.

The nipple is retracted and is connected with the carcinomatous infiltrate.

in front of the clavicle into the supraclavicular nodes; the muscular and deep mammary branches probably drain the pectoral fascia.

The *lymph nodes* are situated along the entire length of the blood vessels in close relationship with the veins, and extend from the base to the apex of the axilla. Posteriorly, they lie between the anterior surface of the scapula and the serratus magnus muscle, and medially along the thoracic wall beneath the origin of the pectoralis major muscle. Long chains of lymph nodes follow the perforating branches of the internal mammary arteries, and others lie between the pectoral muscles.

Denuded of the confusion coincident with an effort to make fine

distinctions which possess no practical value, carcinoma of the breast may be divided into the *soft* carcinoma which is richly cellular, and the *hard* carcinoma which is poor in cellular elements.

Histologically, carcinoma of the breast may be classified as *acinous, tubular, atrophic* and *colloid.*

Acinous carcinoma appears in the form of hard and soft nodules. The latter, however, is the more common and represents the most malignant type of medullary cancer. Its coarse stroma is infiltrated by round cells in the form of epithelial clumps, which bear considerable resemblance to acinous glandular elements. The centers of these clumps soon degenerate, soften, and break through the contiguous structures, forming crater-like ulcers, which are the site of spongy granulations and cellular detritus.

Tubular carcinoma is the most common form of cancer of the breast. It is characterized by proliferation of the epithelial complex in the form of long interlacing cylinders or tubules which soon extend beyond the original acini and finally coalesce with them. Gradually the process progresses toward the surface of the gland and infiltrates the contiguous tissue, forming long tubular cancer nests. At the same time, proliferation of the connective tissue keeps pace with that of the epithelium.

As the cancerous infiltration extends, it invades the skin, the fat, the muscular tissues, the ribs, and the pleura, each of which becomes the seat of cancer nodules. In not a few instances the overlying skin is permeated by numbers of small nodules which extend over the surface of the breast, the back, and the arm (*cancer en cuirasse*) while the skin itself becomes the seat of a hard red infiltrate (Fig. 608).

In tubular infiltrating cancer of the breast, cellular degeneration is much less rapid than in the acinous form. A portion of the detritus is taken up by the veins and the remaining cells are surrounded by proliferated interstitial scar tissue, which is manifested by retraction of the skin and the nipple.

Scirrhus or cicatrizing cancer is characterized by its slow growth, the disappearance of cancer tissue, and the subsequent cicatrical shrinkage and induration of the stroma. Connective tissue predominates and the cancer nests are small and sparse. The microscope reveals masses of elastic fibers flecked with tubular cancer nests. This form of cancer is usually found in elderly women.

Colloid carcinoma is not a frequent visitor in the mammary zone. The colloid constituent is the outcome of mucoid degeneration of the

connective tissue surrounding the cancer nests (Lange[7]) or of the epithelial cancer cells (Ribbert[8]). The degeneration is attended with the disappearance of the cancer cells surrounded by the mucoid substance.

Colloid carcinoma of the breast grows slowly and gives the best operative prognosis.

On *section,* carcinoma is revealed to the naked eye as a grayish

FIG. 869.— MEDULLARY CARCINOMA OF BREAST.

red or grayish white mass containing a network of connective tissue strands in which a softer tissue is embedded. It is not encapsulated and merges into the surrounding tissues either as a nodule or as a more or less diffuse infiltrate. The so-called cancer juice may be expressed from the cut surface. Microscopically, this fluid is found to consist of the detritus of large epithelial cells which contain nuclei and glistening nucleoli.

The *etiology* of cancer of the breast is fully discussed in connection

with cancer in general (Part VI, chap. xvii). The anatomical distribution of cancer of the breasts accords with the arrangement of its lymphatic connections as stated above.

As carcinoma of the breast, in its early stages, is not attended with pain, it rarely comes under the observation of the surgeon during this period of its development. As a rule the accidental discovery of a nodule first attracts attention. Later in the progress of the disease, *lancinating pain* in the breast radiating to the shoulder and arm makes its appearance. Occasionally the discharge of a yellowish or brownish fluid from the nipple heralds the advent of the affection. However, this also occurs in the course of benign tumors and does not possess diagnostic significance.

Palpation of the breast reveals the presence of a more or less coarse tumor, the surface of which is likely to be nodular or lobulated. The nodule, irrespective of its size, cannot be moved within the breast; this FIXATION OF THE TUMOR IS AN IMPORTANT CRITERION OF ITS MALIGNANT CHARACTER.

The rapidity of the growth of cancer is dependent upon its histological structure. Alveolar carcinoma grows rapidly; scirrhus, very slowly. However, ALL FORMS OF CANCER ARE CHARACTERIZED BY THE UNRESTRICTED INVASION OF CONTIGUOUS TISSUE. It extends to the *skin which becomes adherent to it.* The skin soon presents punctate depressions which give it the appearance of the surface of an orange (*peau d'orange*). This is due to lymph stasis. The promptness with which the skin becomes adherent to the tumor depends upon the location of the latter. At the same time, the cancer extends centrally and invades the pectoral muscles and the ribs. The inability to move the breast upon the pectoral fascia indicates that the tumor has become adherent to it. When coalescence with the ribs has taken place, the breast cannot be moved in any direction.

Retraction of the nipple is regarded as an important indication of cancer. It occurs, however, in all processes involving the breast attended with shrinkage of its stroma. It is, therefore, most marked in cases of scirrhus carcinoma, but is present in interstitial mastitis, healed abscesses, etc.

Enlargement of the axillary lymphatics is said to occur late in the disease (usually after from twelve to eighteen months.) This probably refers to the clinical manifestation of glandular involvement. It is the experience of all surgeons that glandular invasion is present in cases operated upon long before the glands are palpable through the

skin. Carcinomatous glands are hard, coarse, and not tender. Pain in the arm indicates invasion of the axillary lymph nodes, which are making pressure on the nerve trunks. The gland located at the edge of the pectoralis major muscle, level with the third rib, is the first to be involved. From this the glandular invasion extends in the form of a wreath high up into the apex of the axilla. Later, the cervical and clavicular nodes are invaded. The extent of the glandular involvement is in accord with the virulence of the cancerous process.

As a rule, cancer appears as a single nodule. Multiplicity of primary lesions indicates virulence of the process. Simultaneous invasion of both mammae is of exceedingly unfavorable prognostic significance.

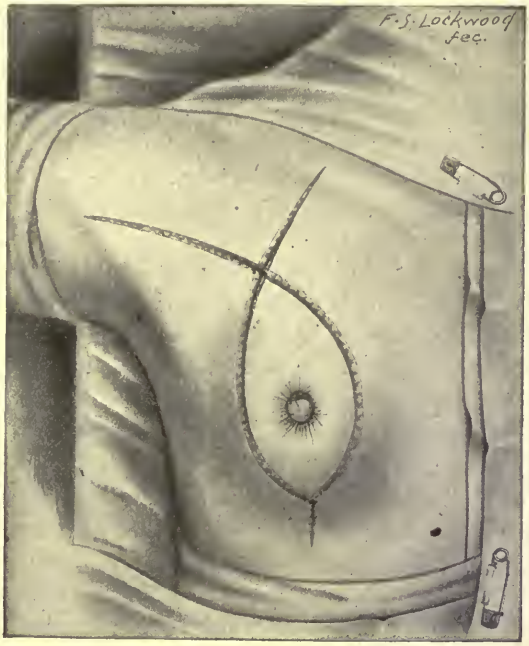

FIG. 870.— AMPUTATION OF BREAST: SKIN INCISION.

A remarkable accord in statistics shows that IN THE MAJORITY OF CASES CANCER BEGINS IN THE OUTER, UPPER QUADRANT OF THE BREAST—58 per cent as against 18 per cent in the other sectors (v. Angerer[9]).

The *diagnosis* of cancer of the breast is first concerned in differentiating a benign from a malignant tumor. On the basis of experience one is justified in assuming that a tumor appearing in the breast of a woman more than forty years of age is likely to be malignant in character. In cases presenting a clearly defined clinical picture the diagnosis is not difficult. When, however, this picture is presented, eradication of the disease, even by the most radical operative measures, is, to say the least, doubtful. The PRESENCE OF ANY ONE OF THE

SIGNS OR SYMPTOMS STATED ABOVE JUSTIFIES radical measures of relief. The value of the serological test is not yet shown.

The *treatment* of cancer of the breast is clearly indicated in connection with the discussion of cancer in general (Part VI, chap. xvii). It is perhaps justifiable to state that in no portion of the body is it more important that radical operative measures be employed. The

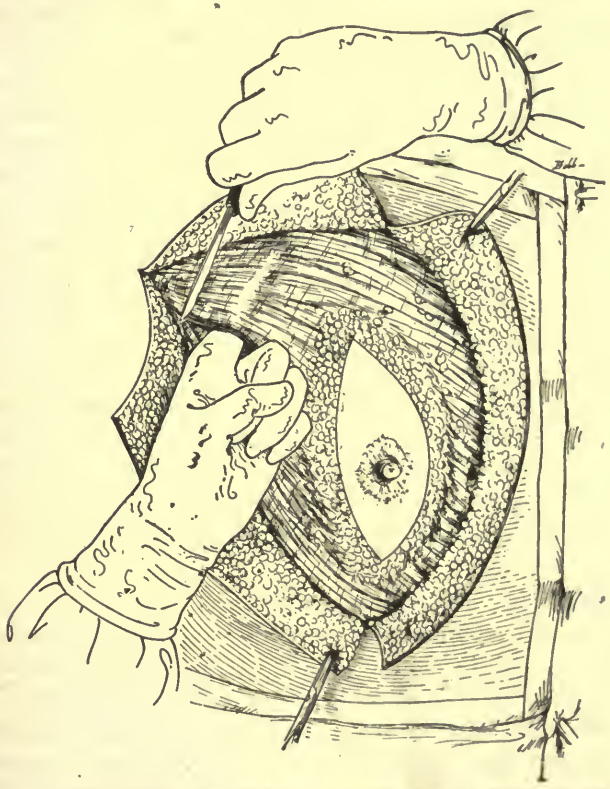

FIG. 871.—AMPUTATION OF BREAST: SECTION OF PECTORALIS MAJOR.

methods of procedure are numerous, yet, when analyzed, have much in common. The following, quoted from Finney,[4] would seem to convey an adequate notion of the operative procedure indicated in this class of cases:

"The most important point to be considered in the choice of incision is that it should include enough skin. Any one of several classical incisions will be found satisfactory. It should be made to conform

to the location of the growth. The amount to be removed having been outlined as shown in Fig. 870, the next important step is the dissection of the skin flaps; beginning with the upper outer one, these should be reflected all around the area of skin to be removed, leaving exposed the fat covering the breast and the axillary structures as far back as the free border of the latissimus dorsi, the posterior boundary of the axilla. Care should be taken to remove every vestige of breast tissue; in other words, one should not see breast tissue until it has been removed.

"The next step in the operation is the division of the pectoralis major close to its insertion into the humerus. It should be divided on the finger from without inward (Fig. 871). The divided muscle is then retracted downward and inward, and the upper part of the pectoralis minor, covered with its overlying fascia, then comes into view. This also is divided on the finger. The axillary vessels and nerve trunks are now exposed by dividing the overlying fascia. The incision of the fascia is carried along the vessels up to the clavicle, where there come into view the uppermost fibers of the clavicular portion of the pectoralis major. These are freed and divided. The fingers of the left hand are now inserted from above downward between the remaining fibers of the pectoralis major and the chest wall. The muscular fibers are thus lifted from the chest and made tense, rendering their division on the finger easy. Just enough margin of the muscle should be left to secure the bleeding vessels, which can be seen and clamped before they are cut.

"The breast and divided pectoralis major now drop downward, exposing the origin of the minor, which is divided in a similar manner. Beginning now at the lower border of the clavicle, the vessels and nerve trunks are cleared absolutely free of all fat, lymphatic and other tissues, from above downward. The vessels and nerves are thus fully exposed in the apex of the axilla. The dissection is carried down along the vessels as far as the outer border of the axilla; the various branches, both venous and arterial, being exposed, are divided between two clamps. The axillary fat, lymphatic vessels and glands — involved and uninvolved — the vascular attachments having been divided, can now be readily separated from the axillary artery and vein, and from the scapular muscles and chest wall, being held by loose areolar tissues only. The breast, together with its overlying skin and fat, the pectoral muscles and fascia, the axillary glands, lymphatics and smaller vessels and nerves with their surrounding fat, after dividing the subcutaneous

tissue along the free border of the latissimus dorsi, are now removed in one piece.

"Aside from the brachial plexus, the only nerves which need be saved are the subscapular. They should be sacrificed rather than run the slightest risk of leaving infected tissue. If the proper amount of skin has been removed, it will generally be found impossible to completely close the wound. After replacing the skin flaps as advantageously as posible, the remaining portion of the wound should be immediately covered with skin grafts after the method of Thiersch. If, in the reconstruction of the axilla, sufficient care is exercised in keeping the axillary skin flap tucked well up into the apex of the axilla by means of properly applied pads of gauze, the subsequent usefulness of the arm will be found little impaired."

Of the other methods of making the incision, those of Warren,[10] Halsted,[11] and Franke[12] are perhaps most frequently employed.

The removal of benign tumors is accomplished through atypical methods of excision. In a certain number of cases it is feasible to elevate the breast through an incision made along its inferior border, the growth being removed from the posterior surface of the organ. When this is accomplished, the breast is allowed to fall into place and the wound is sutured. Drainage is usually necessary.

The so-called *ray treatment* and the *palliative measures of relief* employed in connection with carcinoma are taken up in Part VI. The beneficial effect of *oöphorectomy* (Beatson[13]) is doubtful.

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PART XVI

THE SURGERY OF THE ABDOMEN

CHAPTER I

SURGERY OF THE ABDOMINAL WALL

In injuries and diseases of the abdomen, the abdominal wall plays a relatively minor part. The dangers in this connection relate chiefly to the contents of the abdominal cavity. On the other hand, the abdominal wall presents an anatomical condition of affairs exceedingly favorable to the development of tumors, a fact easily understood from an embryological standpoint. This is especially true of the umbilicus, with which all the structures and organs of this region are connected at some time during the course of the development of the body.

Injuries of the Abdominal Wall.— Subcutaneous contusions and lacerations of the abdominal wall vary in degree. The formation of a large hematoma indicates injury to one of the large vessels (such as the epigastric artery) or to several of the smaller ones. Injury of the vessels of the subserous tissue is attended with the formation of a large hematoma.

Shock, in injuries restricted to the abdominal wall, is usually slight. SEVERE AND PROTRACTED SHOCK AND RIGIDITY OF THE ABDOMINAL WALL INDICATE INJURY TO THE ABDOMINAL CONTENTS, ESPECIALLY TO THE GASTRO-INTESTINAL CANAL. This is equally true when the provocative force is comparatively slight. The possibility of late necrosis of the gut, following injury to the abdominal wall, must be borne in mind.

Cases of injury to the abdomen are always a source of anxiety to the surgeon and require careful study with the view of determining the advisability of surgical efforts at relief. It seems unnecessary to add that, in the event of doubt, exploration is justified.

Subcutaneous laceration of the musculature of the abdominal wall, the outcome of muscular contraction, is not rare. This is likely to happen in acrobats and has occurred in persons convalescing from prolonged infectious diseases (typhoid fever) in connection with slight exertions. One of the recti muscles is usually torn.

The *treatment* is at first directed toward the relief of shock. Abso-

lute rest is imperative. In doubtful cases, a small incision in the abdominal wall clears up the diagnosis. Extensive subcutaneous lacerations of the musculature should be subjected to primary repair by suture. *Hernia* is a frequent sequel following injuries of this sort and is often attended with *strangulation* of the prolapsed gut.

Open wounds of the abdominal wall are divided into *penetrating* and *non-penetrating*. The former are subdivided into *perforating* and *non-perforating*. IN ALL DOUBTFUL CASES THE WOUND SHOULD BE IMMEDIATELY ENLARGED AND THE EXACT CONDITIONS IN THIS CONNECTION ESTABLISHED. UNDER NO CIRCUMSTANCES IS IT PERMISSIBLE TO SEARCH THE WOUND WITH THE FINGERS OR WITH INSTRUMENTS UNTIL ASEPSIS IS ASSURED AND REPAIR OF THE LESION FEASIBLE.

Gunshot wounds are always penetrating. In *stab* and *incised wounds* the omentum or a portion of the gut is likely to protrude. In these cases the deep epigastric artery is often injured.

The *treatment* of non-penetrating wounds of the abdominal wall consists in repair by suture whenever feasible. This is especially necessary in order to obviate the *subsequent development of ventral hernia*. The latter possibility renders the occurrence of infection particularly unfortunate — a reason for the exercise of great care in the toilet of a wound and in its repair. As soon as inflammation makes its appearance, the sutures are removed and the abdomen is kept as quiet as possible by means of appropriate immobilization dressing. In wounds attended with loss of substance, early plastic repair tends to obviate subsequent hernial protrusion.

Diseases of the Abdominal Wall.— Of the *inflammatory diseases* of the abdominal wall, only those *deeply located in the median line possess regional peculiarities.*

Inflammatory lesions beneath the *anterior sheath of the rectus* muscle cannot extend laterally and are arrested in the region of the umbilicus by the inscription tendon. Similar processes *located in the cellular tissue beneath the navel* and between the musculature and the peritoneum of necessity extend to the *prevesical space.*

ACTINOMYCOSIS OF THE ABDOMINAL WALL.— In the vast majority of cases, actinomycosis of the abdominal wall is secondary to that of the intestinal tract.

The *course* of the disease is that of a chronic inflammatory process, occasionally fulminated into activity by an added mixed infection. It has been observed in connection with actinomycosis of the cecum and appendix.

The *diagnosis* is made with the microscope. Clinically, the disease is not differentiated from tuberculosis. For discussion of the treatment see Part II, chap. xix.

Tumors of the Abdominal Wall.— Of the *connective tissue tumors*, *fibromata mollusca* and *sarcomata* are the most common. The former appear as a part of the multiple lesions described in Part VI, chap. xi.

Cutaneous sarcomata develop in connection with *fibromata* and *nevus pigmentosus;* in the latter instance, melanosarcoma is the rule. These extend very rapidly and consequently present unfavorable prognoses. Tumors of this sort should be promptly *extirpated.*

Only two forms of tumors develop from the deeper layers of the abdominal wall, namely, *lipomata* and *desmoids.*

Lipomata are usually subcutaneous, although they also arise from the deeper structures; carcinomatous involution is not rare.

The subcutaneous form of lipomata is usually located at or near the umbilicus, where they emerge through small apertures in the fascia and become subcutaneous. This makes them difficult to differentiate from omental hernia in this situation.

Desmoids of the abdominal wall possess special interest. They arise from the tendinous tissue or from the inscriptiones tendineae of the rectus muscle and are very vascular. A certain number of the tumors become sarcomatous (Stubenrauch[1]). A traumatic origin is claimed but is not proved.

Desmoids are usually located in the median line of the abdomen, where they appear in the form of small, hard, slowly growing tumors. Discomfort is not experienced until the growths reach the size of a hen's egg, when pain is likely to be severe. In the course of a year or more the growth may become as large as a child's head. During the development of the tumor the superimposed skin remains normal except for the presence of enlarged veins.

The *diagnosis* of the presence of a *benign growth* is all that is justified until the exact nature of the tumor is determined by the operative and microscopical findings. The tumor possesses the characteristic of escaping the palpating hand when the abdominal wall is made tense, which, however, only differentiates the mass from an intra-abdominal growth.

The *treatment* consists in extirpation of the tumor. Recurrence is unlikely, though bleeding is often troublesome. Its control requires the patient application of general prinicples. The defect in the abdominal wall may have to be closed by muscleplasty (Kramer[2]).

Of the *epithelial tumors, dermoids* occur at the umbilicus exclusively. *Atheromata* appear in all the sectors of this part of the body.

Primary carcinoma of the abdominal wall (except at the navel) belongs to the surgical rarities. On the other hand, *secondary lympho-carcinomata,* in connection with carcinomata of the abdominal contents, are very common. Mention may here be made of so-called *implantation carcinoma* developing sequentially to operative removal of carcinoma from within the abdomen. Invasion of the abdominal wall by extension from within the abdomen occurs most frequently in connection with carcinoma of the stomach and cecum.

Echinococcus of the abdominal wall is exceedingly rare (Kulenkampff[3]).

Arrest of development of the vitelline duct has already been discussed.

Vitello-intestinal fistula and diverticulum of the umbilical vitelline duct are of practical importance.

A *vitello-intestinal fistula* becomes apparent when the cord separates. It appears in the form of a small red protrusion covered with mucous membrane which exudes mucus or permits of the egress of feces. A probe may be introduced into the gut. When only mucus is secreted, the fistula resembles that leading to an unobliterated urachus. In this event, chemical identification of the constituents of the expelled fluid makes the diagnosis (Lexer[4]). Operative relief is not difficult and consists in excision of the fistulous tract. However, in very young children, the operation should, if the tiny patient's general condition is not unfavorably influenced by the aperture, be postponed until after the second year of life.

Diverticulum of the vitello-intestinal duct appears at the navel in the form of a small pedunculated red tumor of the size of a raspberry. It is covered with mucosa which merges with the normal skin. Removal of the diverticulum opens the peritoneum and should be executed with due consideration for this contingency.

Malformations of the urachus are also described in the general part of this book.

Umbilical fistulae of the urachus are attended with more or less urinary disturbances. They may, as a rule, be extirpated without difficulty. However, the connection with the bladder must be carefully sutured. In a case observed by the writer, the fistulous tract became infected in adult life. Removal of the tract and repair of the

bladder was followed by relief from urinary symptoms which had existed since childhood.

INFLAMMATIONS OF THE NAVEL.— Lack of cleanliness is responsible for most of the inflammatory conditions of the navel. Its funnel shape favors the collection of secretions and foreign substances, which occasionally leads to the formation of so-called "navel stones." These conditions provoke a state of chronic irritation which favors infection and the latter results in dermatitis, cellulitis, and abscess formation. Unless external egress of the exudate is provided, the peritoneum is likely to be invaded.

Inflammatory fistulae of the navel are the result of the extension of similar processes from the abdominal organs. The causative condition may be obscure and finally presents itself at the least resistant portion of the abdominal wall — the navel — in the guise of an acute or subacute inflammation. When the process breaks down, a fistula is formed, the character of which depends upon the location of the underlying process. Thus either a *fecal, urinary, biliary,* or a *simple peritoneal fistula* is formed.

The *diagnosis* is not always easily established. While, in a certain number of the cases, the nature of the discharge is characteristic, in others a conclusion can be arrived at only after careful chemical and microscopical examination of the secretion. The *outcome* of umbilical fistulae depends upon the underlying cause. A number of them heal spontaneously; in others, it is necessary to correct the cause by more or less extensive surgical procedures addressed to the particular condition.

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CHAPTER II

SURGERY OF THE PERITONEUM

Injuries and diseases of the peritoneum are intimately associated with lesions of the organs it covers. Isolated so-called idiopathic diseases and injuries restricted to the peritoneum are exceedingly uncommon. On the other hand, the invasion of the peritoneal covering of an organ by extension from the latter immediately changes the entire aspect of the clinical picture and quite dominates it. When this occurs the surgeon is confronted with a condition involving the largest serous surface in the body and also affecting the organs it mantles.

In diseases of the peritoneum *three important factors* must be taken into account, (1) the *great extent of the membrane* and the rapidity with which inflammations spread over it; (2) its *great capacity for absorption*, which renders it capable on the one hand, of obviating, to a certain extent, the pernicious effects of infections, and on the other, when this is exceeded, of making the consequent toxemia the more menacing; (3) its immediate *response to irritation* by the formation of *an exudate*. The latter may be fibrinous, which leads to the formation of adhesions, or it may consist of a fluid varying in character from serum to pus.

Of the anatomical considerations, those relating to the physiological peritoneal folds and fossae which are often the seat of strangulation are of surgical importance, i. e.,

1. The foramen of Winslow, the entrance to the omental bursa.
2. The intersigmoid recess at the line of origin of the left layer of the mesocolon.
3. The pericecal fossae, i. e., the anterior ileocecal, the ileo-appendicular, and the retrocecal.
4. The duodenojejunal recess (Trietz[1]), sinister sinus venosus (Brösicke[2])—so important in connection with gastro-enterostomy.

Peritonitis.— Inflammations are the most frequent pathological processes involving the peritoneum. On a *pathological basis,* peritonitis may be divided into fibrinous, serous, purulent and putrefactive; from

an *etiological standpoint,* into infectious and non-infectious; *clinically,* into acute and chronic; in accord with its *extent,* into circumscribed or diffuse.

To the surgeon the last division is of especial importance with regard to prognosis and treatment. The clinical picture permits of a clear distinction in this connection. On the other hand, there is no doubt that circumscribed peritonitis is often primarily more or less diffuse in character and is isolated at a certain sector in the abdomen as the outcome of the resistance of the peritoneum. While this somewhat clouds the problem, it is, nevertheless, permissible from a purely clinical standpoint to speak of diffuse and encapsulated peritonitis (Körte[3]).

In by far the greatest number of instances, peritonitis is the outcome of EXTENSION OF INFLAMMATION FROM AN ORGAN WITH WHICH THIS MEMBRANE IS IN CONTACT. *Hematogenous peritonitis* is exceedingly rare. So-called *idiopathic peritonitis* probably does not exist. It is true that there is a form of chronic peritonitis which cannot be definitely connected with a causative lesion. However, this does not exclude its presence.

DIFFUSE PERITONITIS.—Diffuse free peritonitis is characterized by the pathological changes common to inflammations of all serous membranes (Part II, chap. x). Etiologically, it may be divided into *infectious*—the outcome of the invasion of bacterins and toxins, and *non-infectious* (aseptic)—the result of mechanical or chemical irritation.

In the *infectious form* the bacterium coli is the responsible agent in 60 per cent of the cases. Almost all of the bacteria, including the gonococcus, have been found in the peritoneum. As a rule, bacterial peritonitis is not attended with bacteriemia. When it extends, it does so by way of the lymphatics. The bacterial tolerance of the peritoneum is ten times that of the blood; its bactericidal activity is exhausted in six hours.

Aseptic peritonitis occurs in connection with rupture of sterile *cysts* (echinococcus and ovarian), contamination with sterile *bile, urine* and *antiseptics.* The introduction of air is, at times, followed by chronic peritonitis. Contusions are followed by a similar process.

Peritonitis has its origin in all the organs covered by the peritoneum or in contact with it. Of these, the *gastro-intestinal canal* and the *female pelvic organs* are the most common sources of infection.

The active part that the gastro-intestinal canal plays in peritonitis is ascribable to its bacterial flora. Cushing[3] has shown that the ileum is

most productive in this connection. The gastric contents is the least so. *Ulcerative perforation* is the most frequent provocative factor. This may be peptic, typhoid, tuberculous, carcinomatous or luetic. The more acute the perforation, the more severe is the peritonitis. In slowly perforating ulcers the general peritoneum may be protected by adhesions. *Traumatic perforations* of the gastro-intestinal canal produce a condition of affairs similar to the acute ulcerative form. *Phlegmonous processes* of the gut may produce peritonitis by transmigration of bacteria without perforation. The *appendix vermiformis* is responsible for the largest number of infections of the peritoneum of the bacterial type; *peptic ulcers* of the *stomach* and *duodenum* are next in frequency.

In the *small gut,* typhoid and tuberculous ulcers, and in the *colon,* tuberculous and luetic ulcers, most often lead to perforation. To these may be added perforations occurring in connection with *foreign bodies. Obstruction* of the bowel leads to distention perforation; and *mesenteric thrombosis and strangulation* are followed by gangrenous perforation of the gut and subsequent development of peritonitis.

The *intraperitoneal female organs of generation* are often responsible for peritonitis. This may be the outcome of *puerperal infection,* or of the gonococcus (Wertheim[5]); or the infection may develop in connection with traumatic rupture of the uterus.

Infectious cholecystitis, cholangitis, and abscess of the liver are often responsible, and *necrosis of the pancreas* is occasionally responsible for peritonitis. To these may be added the less frequent factors: *rupture of an infected spleen; infections of the mesenteric lymph nodes, of the navel of the newly born, of the prostate* and, by extension, of the *pleura and pericardium.*

Hematogenous peritonitis occurs in the course of acute infectious diseases. Of these, so-called influenza would seem to play a more important part than has heretofore been realized.

Of 867 cases of peritonitis autopsied by Grawitz,[6] the cause was not discoverable in 13.

The *symptoms of diffuse extending peritonitis,* notwithstanding its varied causation, present a *constant clinical picture.* In a number of instances, peritonitis is preceded by the symptoms of the underlying cause. In this event, its development is heralded by a stormy exacerbation of the previously existing manifestations of inflammation. In another class of cases the responsible lesion may not have given rise to disturbances.

The symptoms of peritonitis are, as a whole, similar in character, irrespective of the provocative factor, but *vary widely in degree* in accord with the intensity of the *toxemia*. On the other hand, the severity of the symptom complex does not by any means correspond to the extent of the involvement of the peritoneum; a diffuse peritonitis may be expressed by moderate disturbances and *vice versa*.

Diffuse peritonitis is usually ushered in by a *chill* followed by great prostration. The facial expression is anxious; the nose becomes prominent, while the other features "collapse," especially the eyes, which appear sunken. The *sensorium* is at first clear; later it is clouded (toxemia); pain ceases and cyanosis appears; the pulse becomes thready, the extremities, cool, and the heart, fluttering.

The voice is low pitched; the patient avoids moving the diaphragm, so that respiration is costal and consequently increased in frequency. This is contributed to by the meteoric distention of the gut. The heart action is rapid. The pulse, however (even at first), is small and of low tension. These disturbances are due to vasomotor paralysis of the cardiac and respiratory centers, the outcome of the toxemia (Heinecke[7]).

The *temperature*, when peritonitis develops in the course of a general septic process, is high. In many instances, however, especially in perforation of the appendix, the *temperature is not elevated*. In the latter class of cases, the difference between the axillary and rectal temperature is greater than is normal.

The *functions of the gastro-intestinal canal* are entirely abolished; the stomach even rejects small amounts of fluids. The *tongue* is thickly coated. The *urine* is scanty and contains albumin and indican; *acute hemorrhagic nephritis* is common. On the whole, the patient presents the picture of a severe general infection plus abdominal symptoms.

The *local symptoms of peritonitis* are modified by the organ from which the inflammation emanates. Those *directly* caused by the process are *pain and the formation of exudate*.

Abdominal pain is almost always present and is likely to be severe; in some instances it is sufficiently so to cause shock. In many cases it begins at the location of the primary lesion, for instance, over the site of a perforated gastric ulcer, the appendix, or the female pelvic organs. A conclusion in this regard is permissible only early in the development of the process; later all localizing symptoms are effaced. In the early stages pain is *sharp and lancinating* in character. Later it becomes dull and heavy. *Tenderness* is always present.

The irritation of the inflamed serosa provokes *reflex tension of the abdominal muscles* (rigidity). This is an early symptom of peritonitis and is caused by reflex stimulation of the intercostal nerves. In the beginning of the disease the abdominal wall is retracted, especially in cases of perforation of the gastro-intestinal canal. As a rule, rigidity is greatest over the organ from which the inflammatory process emanates.

The *formation of exudate* varies in quantity and character. In a general way, it may be said that, the stormier the onset of the disease and the more it is associated with acute general infection, the less severe is the local reaction. In these cases the patient is overwhelmed by the disease before the peritoneum has time to react. In less acute cases the peritoneum gives off a purulent or putrefactive exudate and is covered with more or less fibrinous deposits, which collect upon its visceral and parietal layers.

As the quantity of the exudate varies widely, the clinical establishment of its presence is not always feasible. *Modification in the percussion note* is not always indicative in this regard; it may be taken into account in connection with the other symptoms. There is rarely sufficient fluid present to give *fluctuation*. *Diagnostic puncture* is not permissible except, perhaps, in the upper segments of the abdomen when a localized process is clearly defined.

The presence of *free gas* in the abdomen is due to rupture of a hollow viscus or to the decomposition of exudate. The latter is rare. The distention of the gut makes it difficult to be certain that the gas is free in the abdomen.

Pain, the formation of exudate, and abdominal tension are direct symptoms of peritonitis; *vomiting and ileus* are the indirect.

Vomiting is a cardinal symptom of peritonitis and is rarely absent. It begins early in the disease and is maintained throughout its course. While it presents the progressive stages of intestinal obstruction, it is due to reflex causes. The singultus of the later stages is due to reflex irritation of the sensory fibers of the phrenic nerves.

Paralysis of the entire intestinal tract is produced in an analogous manner (reflex irritation of the sensory nerves), so that the contents is no longer propelled. The administration of cathartics does not modify this condition. The arrest of the gastro-intestinal contents is followed by its *decomposition* and the formation of gas, which is responsible for the *meteoric distention of the abdomen*. The diaphragm is pushed upward, the anterior abdominal wall protrudes beyond the niveau of

the thorax, and the liver is pressed upward. If loops of gut are forced in front of the liver, the tympanic note in this situation may lead to the erroneous belief that perforation is present. Paralytic ileus of this sort is finally attended with fecal vomiting.

Leukocytosis is almost always present. However, more importance may be attached to an excessive number of disintegrated leukocytes in the blood.

This is the picture of advanced and, perhaps, unmistakable peritonitis. It is exceedingly desirable that the surgeon should recognize the beginning of the inflammation, so that operative measures of relief may be promptly addressed to the problem. Of the symptoms related above, rigidity of the abdominal wall is the only one which appears early and constantly. THEREFORE, IT MAY BE LAID DOWN AS A SURGICAL RULE THAT WHEN RIGIDITY AND TENDERNESS SUDDENLY DEVELOP IN CONNECTION WITH INJURY AND DISEASE OF THE ABDOMEN, AN OPERATIVE EFFORT AT RELIEF IS INDICATED.

Unless the history of the case is indicative of the location of the primary lesion, the important question as to which organ is the source of the inflammatory process is not easily decided. The differential diagnosis between diffuse and encapsulated peritonitis is also made difficult because the beginning of the latter is always attended with general peritoneal irritation. In addition to this, *biliary and renal colic* are often attended with symptoms resembling those of beginning peritonitis.

The greatest difficulty is the resemblance that the symptomatology of *intestinal obstruction and peritonitis* bear to each other. Until mechanical obstruction has led to peritonitis, several distinctive features are of diagnostic importance. In uncomplicated obstruction the abdomen is not tender nor is it tense; on the contrary, one may often observe the vermicular movements of the gut. In mechanical ileus, fever is absent; in peritonitis it is almost always present. Its absence, however, does not in any sense indicate that inflammation is not present. In all cases of a sudden intra-abdominal lesion in the female, ruptured *extra-uterine pregnancy* must be taken into account. Not the least important consideration is that not a few cases of pneumonitis (especially when the pleura is involved) are ushered in by a clinical picture resembling peritonitis.

The *prognosis* in acute purulent peritonitis is always grave. In the presence of purulent or even putrefactive exudate, the outlook is less menacing than when the peritoneum does not react to invasion. Peri-

tonitis occurring in connection with appendicitis is less often fatal than in gastric perforation. Under all conditions the prognosis is more favorable, the earlier the causative factor is attacked and the leakage of infectious material into the peritoneum arrested.

The *symptomatic treatment* of peritonitis is limited to the control of pain and the maintenance of the heart's action. Because of the uncertainty as to the underlying causation, the administration of cathartics is contra-indicated. The use of opiates interferes with a proper interpretation of the clinical picture.

Of the *surgical treatment of peritonitis,* Moynihan[8] says in substance: In cases of acute general peritonitis operation is indicated, firstly, for the purpose of giving vent to the products of inflammation — pus, seropus, and the escaped contents of wounded viscera; secondly, for the purpose of removing or of otherwise dealing with, that organ or viscus from which the inflammation originally started; thirdly, for the purpose of cleansing, so far as is possible, the walls and recesses of the infected cavity; fourthly, for the purpose of emptying, and, if need be, draining, the intestine whenever it is paralyzed in overdistention; fifthly, for the purpose of providing for a further discharge of free drainage (which can only be of a temporary character) or for shutting off by gauze packing the most infected part of the cavity. In no abdominal operations undertaken by the surgeon is the need for speed, combined with aptness, greater than in these. Slow operations mean death from shock; imperfect operations mean death from a continuance of the acute inflammation. The abdomen is opened by a free incision in the middle line. As soon as the peritoneum is incised, the character of the escaping fluid is noted. The fluid in the case of perforated gastric ulcer is generally turbid, abundant, and flakes of food and of lymph are floating in it. When the duodenum is perforated, the fluid may be bile stained. In both, the gas, which is present free in the peritoneal cavity, is odorless. This is of considerable diagnostic importance, for if the gas be of strong odor, perforation in the stomach or duodenum can almost certainly be excluded. If the fluid be dark brown and offensive, a low perforation of the intestine may be suspected. In cases of typhoid fever in which an ulcer perforates, the fluid is turbid, and contains brownish material and particles of food. The character of the fluid is not always such as to give help to the surgeon. Pus may result from a variety of conditions, and its abominable offensiveness, though suggesting a perforation of

the appendix more decidedly than any other lesion, is not pathognomonic.

The hand or three fingers are now introduced within the abdomen with the utmost gentlenes and care. The search is purposeful. The cecum is first sought, in order to ascertain the condition of the appendix and of the cecum itself. If the appendix is gangrenous, it is dealt with at once in the usual manner. If the cecum is found distended, it is clear that the lesion, if of an obstructive character, is in the large intestine. The sigmoid is then sought; if that too is distended, the condition (if the rectum be unobstructed) is one of general paralytic distention of the intestine due to a universal inflammation. If a search has so far proved ineffective and the condition of the patient permits of further examination, a close scrutiny should be made in order to discover any thick localized deposit of lymph. This, in the majority of instances, will lead to the discovery of the source of offense, for the first and instant response of the peritoneum to injury is the abundant outpouring of lymph and fluid. If it be noticed that, though fluid is everywhere present, a thick deposit of lymph is confined to one area alone, in that area the perforation or other source of irritation may confidently be sought.

After the intra-abdominal manipulations are completed, the question of the cleansing of the peritoneal cavity comes under consideration. However, the following would seem to be the practice of the day. If there is little exudate, lavage is omitted. In cases where there are particles of the food, lymph, or a turbid exudate, a second incision is made in what seems the most appropriate place for affording free drainage. Through this incision a piece of large rubber tubing attached to a funnel is introduced, and a free irrigation of hot sterile salt solution is begun. If both renal pouches are affected and a copious deposit of pus is found in them, incisions are made into them from above the iliac crest. Incision and drainage into these places may serve to prevent a subphrenic abscess.

It is said that lavage of the peritoneal cavity is largely futile, because the recesses and complexities of the peritoneum are such that no flushing, however carefully executed, can possibly clean out all the collections. With a supple rubber tube of good diameter, a free flow of saline solution, and easy means of escape by tubes introduced into other incisions, a reasonably complete cleansing seems feasible.

It is the practice of many surgeons to leave as much fluid as possible

in the peritoneal cavity. Whether much or little is left is a matter of indifference, for if much be left, there is easy egress for it within a very short time through the various incisions, each holding its own drainage tube. Rubbing of the peritoneum, when gauze wipes are introduced for mopping purposes, is to be expressly avoided.

As a rule, only the original incision needs to be sutured, the other openings being made of a size no larger than is necesasry for the introduction of the drainage tube. The making of the multiple incisions does not weaken the abdominal wall, as the surgeon is careful to split through the muscles and also to avoid the section of nerves.

Blake[9] lays down the following operative rules:

1. Remove as rapidly as possible through a small incision the origin of the cause of the inflammation.
2. Irrigate the peritoneal cavity with a double irrigator.
3. Drain as little as possible and do not attempt to drain the general peritoneal cavity.

After the lavage has been completed he closes the incision, leaving a small opening for wound drainage. The drain passes through the wound and just enters the abdomen, allowing the excess of the irrigating fluid to escape, thus preventing wound suppuration. If, after irrigating, one can feel assured that there is nothing left in the peritoneum but what can be absorbed, there is no need of drainage. If there is something left which probably will not be absorbed, one should drain, but should drain only down to the doubtful substance.

In all cases of acute peritonitis when distention, even to paralysis of the gut, is present, the question of evacuating the contents of the gut has to be taken into consideration. There is no doubt that the free evacuation of the stagnant gas and fecal material is desirable. The gut is unlikely to regain its power of contraction when distention has exceeded a certain point, and when this power returns there seems to be a return also of the rapidity of absorption of the intestinal contents. The latter are of an intensely toxic character and their entrance into the circulation is soon followed by a fatal outcome. The emptying of the intestine would seem, therefore, to be justifiable, if carried out in the manner described farther on (p. 1941). Emptying of the gut is a simple, speedy, and satisfactory procedure.

Buxton[10] has shown that bacteria, when injected into the peritoneal cavity, even in minute doses, reach the circulation in a few minutes by way of the lymphatics of the diaphragm. If these results be applied to man, then the Fowler position is indicated to avoid a rush

of bacteria toward the diaphragm and lavage of the abdominal cavity is not indicated.

Despite the views of a number of surgeons the writer feels that the employment of drainage should not be omitted.

After the operation the administration of intestinal stimulants is permissible. Of these, pituitrin would seem to possess the greatest value. Frequent gastric lavage and colonic irrigation are routine measures of treatment. Replacement of fluid by hypodermoclysis is indicated.

Circumscribed Peritonitis.— Acute circumscribed peritonitis owes its existence to the tendency the peritoneum possesses to form adhesions. It is always intimately associated with the organ from which it emanates. The character of the inflammation varies in accord with the underlying cause. This aspect of the problem has already been taken up in connection with diffuse peritonitis.

The *clinical symptoms* of circumscribed peritonitis differ from those of the diffuse form. The onset is attended with general peritoneal irritation, radiating pain, vomiting, etc., but these soon disappear and the evidences of localized inflammation become manifest. This is especially indicated by *localized pain, swelling, and tenderness.* The general condition of the patient does not present so increasingly menacing a picture as does the diffuse form of the disease. The pulse is moderately accelerated, while the temperature corresponds to the degree and character of the localized process.

The *course* of circumscribed peritonitis depends upon the virulence of the invading bacteria. In not a small proportion of the cases the process recedes spontaneously, while others go on to abscess formation. The latter may become encapsulated and remain relatively quiescent for long periods of time, surrounded by a thick layer of connective tissue to which the adjacent gut is adherent, and the exact character of the process does not become apparent until the offending organ — such as the appendix, a fallopian tube, or ovary — is uncovered by the surgeon. As long as the infected organ remains *in situ* the process is intermittently accentuated, so that either the local condition gradually extends, or a sudden rupture of the limiting adhesions is followed by the diffuse form of peritonitis with its dire consequences.

The *surgical treatment* of localized collections of pus in the abdomen is, in a general way, that addressed to the causative organ. The question as to when the attack should be made may be answered here by the statement that the employment of operative measures of relief is

indicated as soon as the presence of pus is determined, provided this can be done without contamination of the contiguous healthy peritoneum.

The presence of pus can be established with certainty only when the focus has reached given dimensions. This is made possible by the symptoms stated above plus a careful examination of the abdomen and due consideration of the pulse, temperature and blood findings. The limitations of diagnostic puncture have already been stated.

The opening should, if feasible, be made within the area of the limiting adhesions. However, experience shows that the removal of pus or of an infected organ through the normal peritoneum is not followed by serious consequences, if the latter is protected from contamination by means of gauze packing. On the other hand, the retroperitoneal approach to pus in the iliac fossa is advisable, especially in pericecal and postuterine suppurative conditions where the contingencies of the given problem may make it advisable to afford egress to the pus at first, and subsequently to extirpate the infected organ.

Of the *special forms of circumscribed peritonitis,* those arising in connection with appendicitis and pyosalpingitis are by far the most common and it is to these two classes of cases that the principles laid down above are especially applicable.

Subphrenic Abscess.— Subphrenic abscess occupies a position by itself. The lesion is located in the *diaphragmatic fossa,* and, as it originates from a number of organs, its recognition is attended with considerable difficulty.

The transverse colon with its dependent omentum forms a sort of partition between the upper and lower segments of the abdominal cavity. Intraperitoneal inflammations are likely to be attended with the formation of adhesions at the site of this division, so that the lower sector is more or less isolated from the upper, which is occupied by the liver, the stomach, the pancreas, and the spleen. Therefore, infections in the last mentioned organs are likely to be the cause of the formation of subphrenic abscesses. To this may be added that the subphrenic abscess may also emanate from an infection of the appendix or be the outcome of diffuse peritonitis.

The subdiaphragmatic region of the abdomen is divided into two sectors by the suspensory ligament of the liver. Right subphrenic abscess usually develops from infections of the *appendix,* the *liver,* the *gallbladder,* or the *right kidney;* the left, from the *stomach, duodenum, pancreas, spleen, left kidney,* and *left lobe of the liver.*

In either event, the lymphatics which perforate the diaphragm may convey the infection to the pleura and cause the development of empyema. Each half of the diaphragm possesses an independent lymphatic system (Küttner[11]).

The *causation* of subphrenic abscess may be stated as follows:

The *appendix* is responsible for subphrenic abscess in two ways: Either the infection is *intraperitoneal,* when it travels along the colon to the edge of the liver and then behind the liver to the under surface of the diaphragm; or it proceeds upward between the layers of the mesocolon and extends to the subdiaphragmatic region by means of the retrocolic connective tissue.

From the *liver and biliary passages* the abscess follows perforation of the gallbladder, the infection extending directly into the subdiaphragmatic space; or the infection may originate from the biliary ducts (*cholangeitis*), an *abscess* of the liver, or an infected echinococcus cyst and extend to the subphrenic fossa.

Slowly perforating *ulcers of the stomach and upper duodenum* are attended with the formation of adhesions between these organs, the liver, abdominal wall, omentum, and spleen, which favor the development of subphrenic abscess on the left side. Rapidly perforating ulcers are followed by diffuse peritonitis. Ulcers of the pylorus and duodenum may also cause the formation of an abscess on the right side.

Purulent inflammatory processes in the lower portion of the *esophagus* may provoke left sided subphrenic abscess.

In addition to this, subphrenic abscess on either side may develop in connection with *empyema,* infections of the *kidney,* or *pancreas,* and perforation of the *transverse colon.*

The *presence of gas* in subphrenic abscesses is due either to leakage from a perforated hollow viscus, or it develops as the result of decomposition of the inflammatory exudate (*pyopneumothorax subphrenicus* of Leyden[12]).

The *clinical manifestations* of subphrenic abscess are not distinctive. They may be sumarized as those of an acute infectious disease with pain and tenderness in the upper abdomen.

The *diagnosis* rests most heavily on the *history,* the *physical signs,* and *aspiration.*

The history reveals the existence of the *previous underlying inflammatory cause* (appendicitis, cholecystitis).

The *physical examination* demonstrates displacement of the organs situated near the diaphragm. *On the right side* the liver is pushed

downward, the diaphragm upward. This increases the area of liver dullness. If gas is present, the upper zone of this area is tympanitic. This three-phase percussion note (lung, air, liver) is characteristic. From behind, persistence of respiratory murmur to the normal level shows the lesion to be below the diaphragm.

In *left sided subphrenic abscess,* the epigastrium protrudes and is tender, and the displaced left lobe of the liver is clearly palpable. The heart is pushed upward and to the left and the left and posterior sides of the thorax emit a dull percussion note, above which a tympanic note may at times be elicited. The latter varies with the posture of the patient. Splenic dullness merges with that of the abscess. In typical cases, the history and the physical findings lead to the diagnosis. In a number of instances, however, a conclusion is not reached.

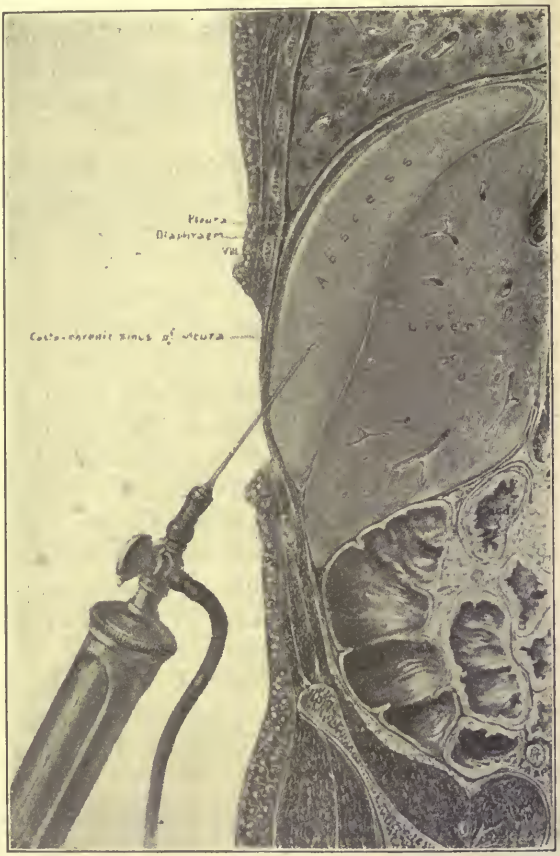

FIG. 872A.—SUBPHRENIC ABSCESS (Kelly).

Aspiration determines the character of the fluid and the depth of its location. The puncture is made over the site of greatest dullness and tenderness and as the needle progresses the piston is gradually withdrawn. Transfixion of the diaphragm is usually conveyed to the hand. The maneuver may have to be repeated several times.

The *outcome* of subphrenic abscess is usually unfavorable unless

operative drainage is employed. Spontaneous discharge of pus through the bronchi may take place, but it is rare. On the other hand, fatal rupture of the abscess into the abdominal cavity, the pleura, or the pericardium is common, while death from general sepsis is still more so.

Opening of the abscess may be accomplished in two ways — transpleural or at the edge of the thorax.

FIG. 872B.— RÖNTGENOGRAM OF SUBPHRENIC ABSCESS: DRAINAGE BY CATHETER.

In the *transpleural route* the aspirating needle is left *in situ* and one or two ribs are resected (usually the eighth or ninth, or both) to the extent of from 6 to 8 cm. The condition of the pleura is now ascertained and if its layers are not adherent by extension of the inflammation from the subphrenic lesion, the operation must be done in *two stages*, the first of which consists in uniting the diaphragmatic to the

costal pleura by sutures or, if these tear out, the area is packed with gauze until the necessary adhesions are formed. In a few days the abscess is opened through the diaphragm.

The *abdominal incision* is made in accord with the location of the abscess and is usually carried along the free border of the ribs. If adhesions have not formed, the general peritoneal cavity is carefully protected by means of gauze pads.

Chronic peritonitis may follow sequentially to an acute process or may be chronic from its onset. It appears in two forms, *chronic exudative,* and *adhesive sclerosing.*

Chronic exudative peritonitis is attended with the formation of a profuse watery exudate and bears a close resemblance to tuberculous peritonitis; it can be differentiated from the latter only by anatomical examination. Not a few observers incline to the belief that the process is, in most instances, tuberculous in origin. Gangitano[13] has attempted to establish a traumatic connection. However, this does not seem to be proved.

The disease is characterized by the gradual accumulation of fluid in the abdominal cavity and the formation of nodular tumors of various sizes, the latter being especially noticeable in the region of the navel.

In the *diagnosis* tuberculosis, carcinomatous peritonitis, and ascites from cirrhosis of the liver, must be taken into account.

Symptomatic *treatment* is advised by some of those who have had experience in this connection. On the other hand, Lennander[14] advises celiotomy and lavage of the peritoneum. This may be repeated, if necessary. As the condition may be due to an occult chronic process susceptible of removal, the employment of exploratory celiotomy would seem to be justified (Körte[3]).

Chronic adhesive sclerosing peritonitis is attended with cicatricial *contraction of the peritoneal folds* (mesentery and omentum) and the *formation of adhesions.* It may develop in a restricted area of the peritoneum or involve its entirety, but it shows a predilection for the vicinity of the *female organs of generation,* the *gallbladder,* the *flexures of the colon,* the *posterior peritoneum,* the *root of the mesentery,* the *sigmoid mesentery* and the *omentum.* The process is the outcome of chronic inflammatory thickening of the peritoneum which, like scar tissue elsewhere, undergoes shrinkage. The most serious result of the condition is the *distortion* and consequent *obstruction of the gut* that it causes.

Chronic adhesive sclerosing peritonitis emanates from all those parts in the abdomen which are the seat of acute inflammatory processes, i. e., those that give rise to acute circumscribed peritonitis. In addition to this the condition appears at the site of *trauma*, especially following operative measures. It is also probable that the disease may be chronic from its onset in response to a process of similar nature in the part or organ in which it has its origin. In the latter class of cases, constipation and hernia are the most frequent causative factors, although, no doubt, luetic lesions of the gut are responsible for the formation of peritoneal adhesions in a not inconsiderable number of instances.

Chronic adhesive sclerosing peritonitis does not present a distinctive clinical picture. The *symptoms* develop very gradually and are the outcome of interference with the function of the organs affected by the process — such as evidences of disturbances in the secretion of bile, pyloric obstruction, kinking of the colon and sigmoid, displacement of the uterus, etc. Of the *diagnostic measures*, the X-ray findings are the most illuminating.

The *operative treatment* of the adhesions themselves consists in their division or extirpation. When feasible, the causative organ or part should be removed (cholecystectomy, appendectomy, salpingectomy, etc.).

Tuberculous Peritonitis.— Tuberculosis of the peritoneum appears in two forms: As a part of *miliary tuberculosis*, which possesses no surgical importance, and in the form of a *tuberculous inflammation of the serosa*. In the latter form of the disease the peritoneum is red, swollen and hyperemic, and its surface is rough and covered with numerous gray nodules of varying size. The process is attended with the formation of an *exudate*, which may be fibrinous, serous, purulent or hemorrhagic, or present a combination of the four.

Clinically, tuberculous peritonitis is divided into the following forms:

1. Tuberculous peritonitis with the formation of a copious serous *exudate*.
2. That attended with the formation of adhesions and *nodular tumors* in the peritoneum, mesentery and omentum, and with shrinkage of the peritoneal folds.
3. The ulcerative purulent type, in which the intestinal loops are agglutinated and the collections of exudate are likely to be sacculated.

The various forms often merge into one another.

Tuberculous peritonitis is rarely primary. Of 226 cases autopsied by Borschke,[15] the existence of a focus elsewhere in the body was not demonstrable in two. As a rule, the process is secondary to tuberculosis in the lung, although it may also arise in connection with infection in the gut and genitals.

The *general symptomatology* of tuberculous peritonitis is that of a gradually *increasing cachexia* with low fever and moderate increase in respiration and pulse rate. The last is progressively accelerated. The symptoms of inflammation are not clearly defined.

Tenderness is slight; vomiting does not usually occur; *constipation* is the rule. The latter is due to intestinal stenosis. *Diarrhea* indicates coexisting enteritis. *Distention* and the *collection of fluid* becomes increasingly manifest as the process progresses. In a number of cases nodular thickening is palpable.

The *diagnosis* is made on the symptoms enumerated above. As already stated, the process cannot be diagnosticated clinically from the non-tuberculous form of chronic peritonitis. The Pirquet test and the inoculation of aspirated exudate are of value. Differentiation from carcinomatous peritonitis can be made only after opening the abdomen. *Leukopenia* indicates tuberculosis. Ascites, due to hepatic cirrhosis, must be taken into account.

The outcome of tuberculous peritonitis is not necessarily unfavorable. No doubt a number of cases recover spontaneously. It is also probable that the adhesions discovered during celiotomy are not infrequently remnants of *healed tuberculosis*. Borchgrevink[16] presents an interesting statistical review in this connection.

Aside from general sustaining *treatment, operative measures of relief* would seem to possess therapeutic value. The operation consists in celiotomy and the admission of air into the peritoneal sac. The abdomen is opened a distance of from 8 to 10 cm.; fluid exudate is removed and the peritoneal surfaces are carefully and gently dried, iodoform gauze being used for the purpose. Healing is ascribed to the hyperemia which follows the entrance of air and the intra-abdominal manipulations, i. e., artificial leukocytosis is produced. However, a number of observers do not regard the procedure as justified (Shattuck[17]).

Injuries of the Peritoneum.— Unless attended with *infection*, trauma to the peritoneum is well borne. Infection may be introduced *from without* or, which is more often the case, it gains access to the peritoneum from an *injured viscus*.

Traumatic solution of continuity of intra-abdominal organs, like that due to *ulcerative perforation,* leads to peritonitis. The fact that the former involves a healthy organ, and the possibility of spontaneous closure, makes the outlook somewhat less serious. This is, however, in a measure counterbalanced by the danger of *intra-abdominal bleeding.*

In penetrating abdominal injuries the most IMPORTANT QUESTION IS WHETHER THE GUT OR A BLOOD VESSEL IS INJURED. Unfortunately the abdominal contents rarely escapes, especially in gunshot and stab wounds. In a general way it may be stated that injuries of the upper sectors of the abdomen are most likely to be uncomplicated by visceral involvement.

Internal bleeding is evinced by the clinical picture of hemorrhage and is easily recognized. The establishment of *visceral injury* is more difficult. As a matter of fact, the diagnosis rests entirely on the early symptoms of peritonitis, of which rigidity and distention are the most indicative in this connection.

Much has been said for and against immediate exploration of the extent of injuries of this sort. However, the writer has no hesitation in stating THAT, IN VIEW OF THE DANGERS ARISING FROM INJURIES TO VISCERA AND BLOOD VESSELS, IT IS NECESSARY IN ALL CASES OF TRAUMA TO THE ABDOMEN, THE NATURE OF WHICH AROUSES SUSPICION OF INVOLVEMENT OF THE CONTENTS, THAT THE ABDOMINAL CAVITY BE FREELY OPENED AND THE INJURED AREA EXPOSED, PROVIDED THIS IS DONE UNDER ASEPTIC PRECAUTIONS. While it is true that occasionally an early operation of this sort does not uncover an injured viscus or blood vessel, it is also true that the surgeon is not justified in exposing the patient to the risks that arise in connection pith a policy of expectancy. After the injury is exposed it is repaired on general surgical principles.

Subcutaneous injuries of the abdominal organs, the result of blunt force, present an exceedingly difficult problem. The gut may be torn, but the compact organs — the liver, spleen, pancreas, and kidney — are most likely to be injured. Of the hollow viscera, those containing fluid — the *gallbladder, stomach, and urinary bladder* — are most frequently ruptured. The intestine is most often injured by crushing force. Avulsion of the mesentery is followed by intestinal gangrene.

The *dangers* of subcutaneous injuries lie in hemorrhage, the discharge of *infectious elements* into the abdominal cavity, and in *shock,* which is always severe.

In this class of cases the indications for operation are very indefinite.

If shock is maintained, it is fair to assume that an intra-abdominal injury is present. Identification of the injured organ must rest on a study of the character and location of the injury. In most instances median celiotomy will have to be employed. While the development of the symptoms of peritonitis makes the diagnosis certain, these should not be awaited before surgical effort at relief is made. The rule laid down above in connection with penetrating injuries is equally applicable to subcutaneous abdominal injuries.

The *late complications* of injuries of this sort consist in the formation of *adhesions* (stenosis of the pylorus and gut) and fecal fistulae. The effusion of sterile bile and urine is followed by aseptic chronic peritonitis, with the ultimate possibilities discussed above. Encapsulated bloody effusions (*intraperitoneal blood cysts*), unless opened and drained, are followed by similar events. They are most frequently located in the region of the pancreas and spleen and in the mesentery and omentum (Stimson,[18] Lexer,[19] Tawaststjerna[20]).

Tumors of the Peritoneum, Subperitoneal Tissues, Omentum and Mesentery.— Of the *malignant tumors, colloid carcinoma, endotheliomata and plexiform angiosarcoma* most frequently appear in the peritoneum. *Pseudomyxoma* is a term applied to collections of colloid tumor masses of the peritoneum which are usually secondary to colloid growth of the ovary, gut, or appendix. As a rule, malignant tumors extend to the peritoneum from the abdominal organs in the form of closely strewn nodules, and also usually involve the mesentery and omentum. In most instances the peritoneum also contains a certain amount of *fluid exudate.*

The affection is recognized by the appearance of steadily growing nodules and of a fluid exudate. Evidences of inflammation are very slight or absent and are usually overshadowed by the symptoms emanating from the organ in which the growth has its origin. This is also the deciding factor as regards tuberculous peritonitis.

The *treatment* is purely symptomatic. In several instances the peritoneal process has subsided after removal of the affected organ (Körte[3]).

The *retroperitoneal tissues* are not rarely the seat of lipomata, fibromata, myxomata, and mixed tumors which grow very slowly and may attain considerable size. These tumors extend out between the layers of the mesentery, displace the gut and make pressure upon the contiguous organs, especially the kidneys, ureters, and great vessels.

Tumors of this sort are usually best approached by the *transperitoneal route* through a median celiotory. As a rule, extirpation may be accomplished in this way. However, if the process has its origin in an area which makes this impracticable, the oblique incision for exposure of the kidney must also be employed.

Solid tumors of the omentum are likely to present difficulties. In this class of cases care must be exercised not to damage the transverse colon. In some instances, it will be found necessary to remove a portion of the latter and reëstablish the continuity of the gut by anastomosis.

Inflammatory tumors of the omentum develop sequentially to operations for hernia, removal of the appendix, etc., in which it is necessary to deligate the omentum and remove it to a greater or less extent. In these instances a peculiar inflammatory thickening develops which bears a resemblance to malignant disease. If suppuration does not supervene the process gradually subsides.

Cystic tumors of the omentum, mesentery, and subserous tissues are of varying kinds. *Echinococcus* is not infrequent and is usually secondary to a cyst of the liver or other abdominal organ.

Cysts located between the layers of the mesentery or omentum are *serous* or *chylous* and arise in connection with disturbances of the lymphatic or chyle vessels or are so-called *blood cysts,* the outcome of a hematoma. The cysts often develop to a great size but are usually freely movable and are surrounded by the gut. During operative removal it is important to recognize the point of origin of the cyst, so that the blood supply of the gut will not be impaired. If the purpose cannot be accomplished without endangering the integrity of the gut, it is best to resect a portion of the cyst and fasten the remainder to the abdominal wall. This is followed by gradual obliteration of the cavity.

Teratoid tumors of the abdominal cavity are divided into simple and complicated *dermoid cysts, fetal inclusions,* and *mixed tumors. Simple dermoid cysts* originate in the abdominal furrow and are situated in the greater omentum and mesentery of the small gut. Those arising in connection with the wolffian ducts are located behind the peritoneum. *Complicated dermoids* are connected with the ovary or an undescended testicle and also lie retroperitoneally.

Fetal inclusions are usually located between the layers of the transverse mesocolon or in the bursa epiploica.

Teratoid mixed tumors originate from the three embryonic layers and may be located in any of the sectors of the abdomen.

Actinomycosis of the peritoneum is the result of extension of the actinomyces from the gut. The diagnosis is based on the microscopical findings.

For detailed description of tumors of the peritoneum see Part VI.

Ascites.— The accumulation of serous fluid in the abdominal cavity without an inflammatory cause is the outcome of general (heart, lung) and local circulatory disturbances. Of the local disturbances, obstruction of the portal circulation is the most common. Ascites also occurs in connection with the hydremia of nephritis and cachexia. The fluid is, as a rule, clear; the admixture of blood indicates malignant disease or tuberculosis. Occassionally, the fluid is milky from the addition of fat globules (chylous ascites).

Chylous ascites is divided into the *true* and the *chyliform*, the latter consisting simply in the admixture of finely emulsified fat. *True chylous ascites* is the outcome of obstruction of the chyle vessels by tumors, tuberculosis, entozoa, etc.

Ascites is recognized by dullness in the dependent portion of the abdomen, which varies with changes in the posture of the patient, and by the presence of fluctuation.

The *surgical treatment* of ascites consists in aspiration of the fluid. Recently an effort has been made to relieve certain cases by operation. The procedure is based on the work of Drummond and Morrison[21] (1896) and of Talma[22] (1898), who, in cases of cirrhosis of the liver, produced adhesions between the abdominal organs (omentum, gall-bladder, spleen) with the view of establishing a collateral circulation. Ito and Omi[23] have shown experimentally that it is necessary to cause the development of adhesions between the loops of intestines and the abdominal wall, and that omental fixation alone is not sufficient.

A median celiotomy, 6-8 c.cm. in length, is made and the fluid evacuated; the omentum is fastened to the parietal peritoneum with catgut sutures. The experience of the writer with the so-called Talma-Morrison operation has not been encouraging. However, in a case of cirrhosis of the liver operated upon, the fluid had not returned at the end of eighteen months.

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CHAPTER III

OPERATIONS ON THE STOMACH AND INTESTINES

General Considerations.— While the peritoneum is capable of destroying pathogenic bacteria, this is only true provided the invaders do not exceed certain limits as regards quantity and virulence. When this limit is exceeded the result is perhaps more likely to be fatal than in any other portion of the body. The transudate which forms in connection with all abdominal operations is an exceedingly receptive culture medium and when once fertilized, the absorption capacity of the peritoneum is no longer an asset; its cellular elements soon succumb and a menacing condition of affairs develops. While mechanical and bacterial irritation is not well borne by the peritoneum, this membrance also rebels against chemical insult and should, therefore, not be exposed to contact with *antiseptics*. There is no such thing as a harmless disinfectant as far as the peritoneum is concerned. The epoch making work of Lister did not provide its full measure of benefit in connection with abdominal surgery until the technic of asepsis was developed. In no portion of the body is it so important that the principles discussed in connection with the treatment of wounds be more carefully observed, especially with regard to the prevention of *shock* and the *arrest of hemorrhage*. It must also be remembered that the abdomen and its contents cannot be immobilized nor placed out of function after the operation and that this must be taken into account in the after care.

In all cases an *adequate preparation of the patient* is most necessary. Rest in bed the entire day before the operation is essential. The bowel is emptied by liberal catharsis and soap and water enemas. The condition of the mouth must receive close attention, its cleansing being carried out systematically. The measure is regarded as tending to obviate postoperative parotitis and pneumonitis (Moynihan[1]). The administration of sterile diet for forty-eight hours previous to the operation enormously reduces the bacterial flora of the gastro-intestinal canal (Cushing[2]).

The *skin* is prepared by mechanical cleansing the evening before the operation and then covered with a sterile pad; it is painted with tincture of iodin (5 per cent) immediately before the abdominal wall is sectioned. As soon as the incision is made its edges are covered with folds of gauze held in place by clips (Fig. 873), which prevent protruding abdominal contents from coming in contact with the skin.

The question of local anesthesia is not yet settled. Its advantage lies in inhibiting nerve impulses from reaching the brain (anoci-association of Crile[3]); it does not, however, prevent the objectionable psychic strain of the operation. A judicious combination of the two methods would seem advisable.

After the skin is sectioned the *opening of the abdominal wall* is ac-

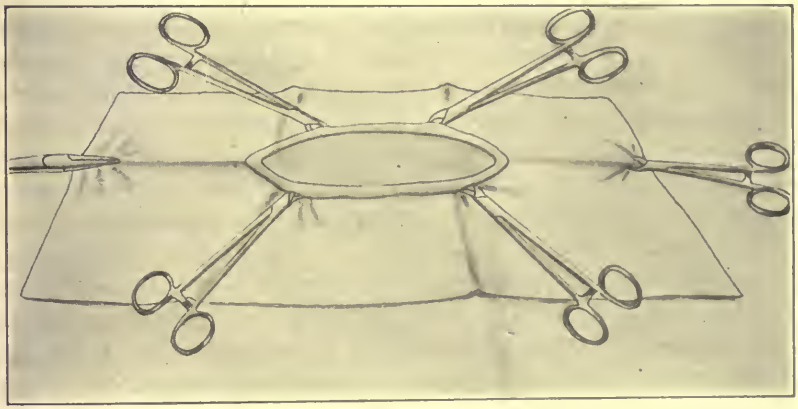


FIG. 873.— GAUZE HELD IN PLACE BY CLIPS.

complished by carefully dividing its various layers, especial care being taken not to injure the gut. The peritoneum is picked up by thumb forceps and incised at the apex of the elevation thus created.

As soon as the *abdomen is opened* an adequate examination of the parts concerned is made. This is necessary, owing to the fact that an exact diagnosis before exploration is often not possible. The lesion having been disclosed, the area to be operated upon is isolated by means of gauze pads, which are made to surround the operative field completely.

Adhesions, the result of inflammatory processes, are frequently found in abdominal operations and may be due to the condition for which the measure is employed, or may have been produced by a healed process of an earlier date. If the adhesions do not interfere with the intent

of the operation and cannot be held responsible for the symptoms presented before the abdomen is open, they need not be disturbed, especially if their separation adds materially to the surgical problem under consideration. However, the question often calls for the exercise of the surgeon's individual judgment.

Separation of adhesions is often attended with difficulties requiring great skill, and, above all, an almost subconscious acquaintance with the resistance of tissues. It is permissible to state that *they should be cut rather than torn.* The less there is of the latter, the less likely are adhesions to form again.

Drainage after abdominal operations has been made the object of much investigation. An extended discussion of the problem cannot be presented here. It is true that drainage agents do not actively remove exudate from the peritoneal sac (which fact has been demonstrated both experimentally and clinically). However, this does not mean that the products of infection are not more likely to find egress through an opening left in the abdominal wall for that purpose than if the wound is tightly closed, especially when there is reason to believe that the formation of exudate will continue after the operation is completed. The limitations of drainage have been carefully studied by Clarke[4] and Yates.[5]

The *after treatment* is an important consideration. As soon as the patient is returned to bed, he is placed in the recumbent position for three or four hours and then propped up in bed. After operations upon the upper sectors of the abdomen, the sitting posture is employed.

Thirst is relieved by the administration of small quantities of water, by enteroclysis, and, if necessary, by hypodermoclysis. After twenty-four hours fluid diet may be ordered. While the use of morphia is objectionable for many reasons, and the drug should be withheld when possible, the clinician will find it necessary to administer it in most instances (1/6 to 1/4 of a grain given hypodermically). *Flatulence,* is combated by the hypodermic injection of *eserin* or *pituitrin,* by the introduction of the rectal tube, and by low enemas.

Complications and Sequelae of Abdominal Operations.— The chief postoperative complications of abdominal operations are (1) peritonitis; (2) lung complications; (3) parotitis; (5) postoperative hematemesis; (5) acute dilatation of the stomach; (6) phlebitis and thrombosis.

1. *Peritonitis* has already been discussesd (p. 1890).

2. *Lung complications* — pneumonitis, pleuritis, bronchitis, edema of the lungs, abscess of the lungs, massive collapse, pulmonary embo-

lism, and gangrene of the lung — have been attributed to the effect of the narcosis. As each of these has followed abdominal operations under local anesthesia, this theory is no longer tenable. After extended discussions, Moynihan,[1] Körte,[6] and W. Pasteur[7] ascribe the occurrence of lung complications to aspiration of foreign materials, the lodgment of emboli from ligated vessels, rapid cooling of the surface of the body, and restriction of respiration because of the abdominal trauma. Lung affections occur most often after operative invasion of the upper sectors of the abdomen (Körte[6]). In view of the prevalent notions of the "fresh air fiend," it would seem permissible to emphasize the influence of refrigeration in this connection. Moynihan[1] warns us that intravenous infusion may be responsible for some of the cases. Bibergeil,[8] after an analysis of 3,909 abdominal sections, of which 139 developed pneumonitis, attaches much importance to the aspiration of infectious material from the mouth.

3. *Parotitis* has been studied by Bucknall,[9] who seems to have shown that the infection extends to the gland from the mouth and that the complication may be obviated by the toilet of the latter.

4. *Hematemesis* after abdominal operation, other than that following operations upon the stomach, has been ascribed to many causes. At this writing there is still a wide diversity of opinion in this connection. No doubt sepsis is often responsible for the condition; on the other hand, capillary oozing following dynamic paralysis of the stomach and gut, the result of trauma, is probably the most important causative factor. This view places the problem in a close relationship to acute dilatation of the stomach and gut.

5. *Acute dilatation of the stomach* is a most serious postoperative complication. While the affection has been most frequently observed in connection with abdominal operations, it also follows operations on other portions of the body and has been observed (by the writer[10]) in connection with brain injuries. The subject has been carefully studied and many theories have been advanced with regard to its causation. Of these the conception that the condition consists in an *atony of the stomach wall* due to paralysis, the outcome of shock, is perhaps the one most widely accepted (v. Eiselsberg,[11] Luckett,[12] Kelling,[13] Thompson[14]).

The *symptoms,* as a rule, appear insidiously. The chief disturbances are pain in the epigastrium, constant effortless vomiting of a thin greenish or grayish tinted fluid, *rapid and steadily increasing pulse rate,* and the clinical picture of collapse and exhaustion. Examination re-

veals a tense distention of the epigastrium, and the inflated stomach may be outlined through the abdominal wall. Upon introduction of the stomach tube gas is explosively expelled, but reaccumulates in a short time. *Respirations* are frequent and shallow; *thirst* is intolerable; the *urine* is scanty. The gross pathological findings are presented in detail by Box and Wallace[15] and by Ordway.[16]

The *treatment* consists in frequent siphonage and lavage of the gastric contents and in the administration of eserin and pituitin.

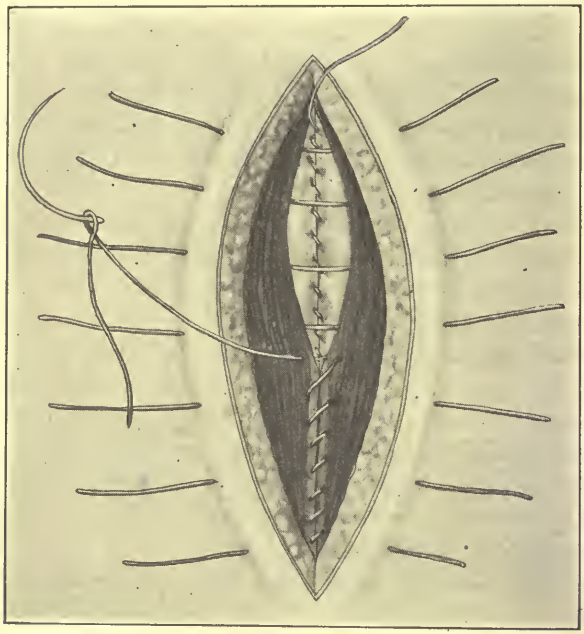


FIG. 874.— SUTURE OF ABDOMINAL WALL.

The continuous suture returning and approximating the sheath of the rectus muscle.

6. *Phlebitis* is ascribable to the absorption of infective substances from the site of the operation. It follows hysterectomy in 30 per cent of the cases. The subject is extensively discussed in Part II, chap. x.

Abdominal Incisions. — In opening the abdomen it is a cardinal rule that free division of muscle fibers and nerves is to be avoided. It is probable that failure to observe this admonition is more often responsible for postoperative hernia than is infection of the wound. This was first brought out by Asmy,[17] whose work led to the employ-

ment of oblique and intermuscular incisions. In no other portion of the body is it so necessary that trauma to the edges of the wound be avoided. This argues for free exposure of the area to be attacked and the avoidance of forcible retraction of the superficial parts.

In the *suture of incisions* great care is necessary. As a rule three layers of sutures are used in the following manner: A round curved needle, armed with a long thread of No. 2 catgut is used. The first stitch is introduced at the upper portion of the wound and transfixes the peritoneum, the fascia transversalis and the posterior sheath of the rectus. This suture is continuous and ends at the lower aspect

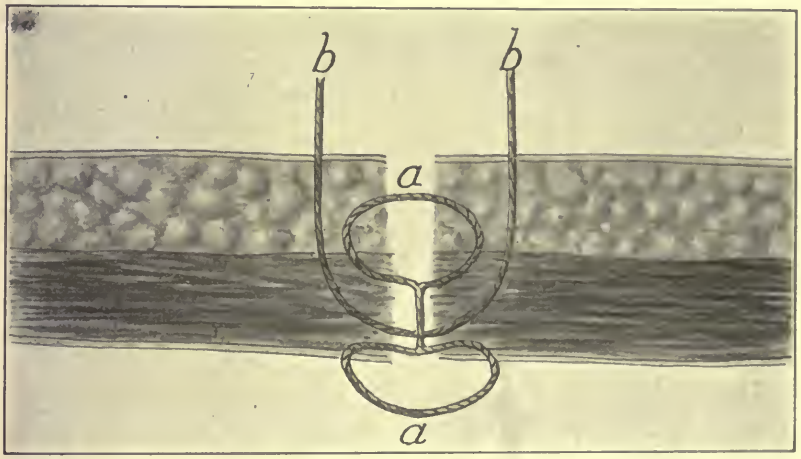


FIG. 875.— SUTURE OF THE ABDOMINAL WALL.

a a Are the different parts of the same continuous suture; *b b*, interrupted sutures.

of the wound. A series of silkworm gut sutures is now passed through all the thicknesses of the abdominal wall not embraced by the first catgut suture. A surgical needle is used for the purpose. These stitches are introduced at intervals of about three fourths of an inch along the full extent of the wound. The ends are left loose on either side and are seized by a clip. The anterior sheath of the rectus and a few of its muscular fibers are apposed with No. 2 chromic gut. The interrupted silkworm gut sutures are now tied. The skin is approximated with a continuous suture of fine silk.

Operations on the Stomach.— For the relief of *gastric perforation*, the abdomen is opened in the middle line above the umbilicus by an

incision of ample size. As soon as the abdomen is opened, gas or a small quantity of thin, clear or turbid fluid escapes. The ulcer is rapidly sought and, together with the necessary amount of stomach wall, is drawn well up into the wound and isolated from the peritoneal sac by means of gauze swabs. The opening is at once closed by a single stitch which passes through all the coats of the stomach on each side of the perforation. This prevents further escape of the contents. The ulcer is not excised, but, together with its surrounding area of

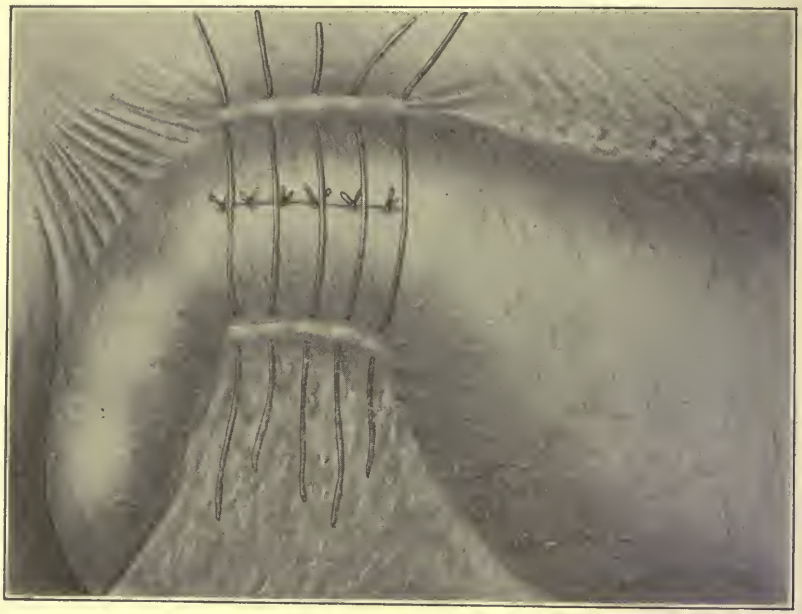


FIG. 876.—REPAIR OF DUODENAL ULCER.

The gastrohepatic and gastrocolic omenta are sutured over the defect.

edema, is turned into the stomach by a double layer of sutures, using fine chromic gut (No. 00) for the purpose. After closing the perforation the toilet of the peritoneum is proceeded with. This consists in carefully wiping out all foreign material with swabs wet with hot saline solution, especial care being taken to cleanse the areas beneath the diaphragm. If the leakage has been excessive and swabbing does not accomplish the purpose, lavage of the peritoneum with saline solution is permissible. Drainage is advisable. The question of immediate gastro-enterostomy in pyloric perforation is an involved one. The

operation is justified when repair of the perforation necessitates occlusion of more than one third of the lumen of the pylorus and the general condition of the patient does not contra-indicate the effort. It should not be practiced as a routine measure, despite the fact that not a few cases of this sort must ultimately be subjected to the operation.

Perforated Duodenal Ulcer.— Perforated duodenal ulcer possesses certain peculiarities. The leakage escapes upon the upper surface of the transverse mesocolon to the right of the elevation caused by the contact of the transverse colon with the greater curvature of the stomach. It therefore tends to run to the right of the hepatic flexure and then descends along the outer aspect of the ascending colon to the right iliac fossa, where it is likely to provoke the manifestations.

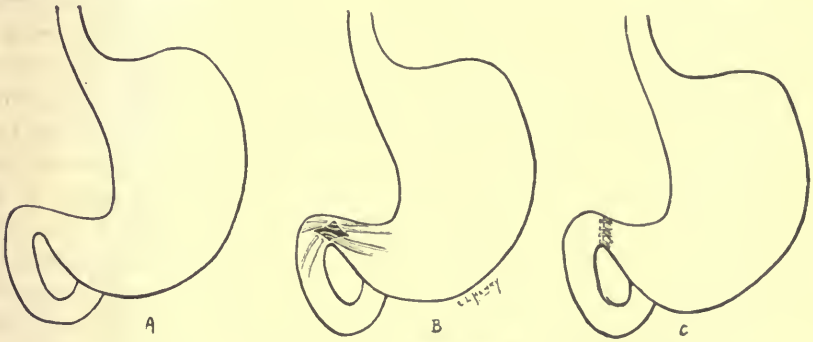


Fig. 877.— Various Steps of Simple Pyloroplasty.

of appendicitis. From this region the fluid, after filling the pelvis, encroaches upon the left iliac fossa.

The *symptoms* of perforated duodenal ulcer do not differ materially from those of gastric perforation, except, perhaps, that the signs in the former are more marked on the right side of the abdomen.

Direct closure of the perforation is feasible only when it is located upon the anterior surface of the duodenum. When it is inaccessible the area may be covered by folding the two layers of omentum over it.

Pyloroplasty.— Pyloroplasty aims at widening a stenosed pylorus. After opening the abdomen through the right rectus muscle, the pylorus is delivered into the wound and incised in its long axis a distance of from 4 to 6 c.cm. The wound is then closed in the same axis (Fig. 877).

Posterior Gastro-enterostomy. Posterior gastro-enterostomy is performed in the following manner:

An incision about 4 inches in length is made about one inch to the right of the middle line, above the umbilicus. The stomach, transverse colon and omentum are withdrawn from the abdomen and turned upward, exposing the under surface of the transverse mesocolon. The origin of the jejunum is then identified. The posterior surface of the stomach is exposed by making an opening in the transverse mesocolon into the lesser sac; a snip of the scissors by the side of a forceps suffices for the purpose. The opening is enlarged by stretching and tearing until three fingers can readily be passed through it. Through this, the part selected for the anastomosis is projected by an assistant. A fold of the posterior surface of the projecting portion of the stomach is then seized and drawn well out of the abdomen; as the fold is held in the fingers of the left hand, it is embraced by a clamp applied vertically, the tip of the blades pointing toward the chin, the handle to the pubis. Three to four inches of stomach wall should be included in the grasp of the clamp; the latter is then turned transversely, the handle pointing to the left side of the abdomen, in which position it is held by an assistant. The upper portion of the jejunum, previously isolated, is now drawn well out of the abdomen and clamped to the extent of 3 or 4 inches of the gut. The two clamps now hold the portion of stomach and intestine to be anastomosed. The portions of stomach, omentum and transverse colon delivered throngh the wound are now returned to the abdomen. The two clamps are held apart; a roll of moist, hot gauze is placed between them; over this they are approximated as closely as possible and surrounded by wet pads, so that nothing is visible but the two clamps engaging the viscera about to be united (Fig. 878). The sutures are now introduced; they are all continuous and there is no interruption by knotting at any part of their course; a needle of rather more than half a circle in length and with a rounded body and slot eye, for easy threading, is used, and armed with No. 00 chromic gut. The first stitch transfixes the serous and muscular coats only. It is commenced at the left end of the portions of stomach and jejunum inclosed in the clamps, and is continued until the greater curvature of the stomach, at the right end of the clamp, is reached. The length of the suture line should be from 2 to 3 inches. The individual stitches are placed about one eighth of an inch apart, and the suture is drawn sufficiently tight to easily approximate the serous surfaces. When the first row of sutures is completed, the needle is laid aside, to be used at a later stage of the operation. To either side of this suture line, at a distance of one quarter of an

inch, an incision is now made into the stomach and jejunum, the serous and muscular layers being divided. This causes the mucous membrane to pout into the incisions. An ellipse of the mucous membrane is now excised from both stomach and jejunum, the portion removed being about two inches in length and a half inch wide at the center. It is not usually necessary to ligate any vessels. The inner suture is now introduced; it includes all the coats of the stomach and jejunum around the whole circumference of the opening. The needle

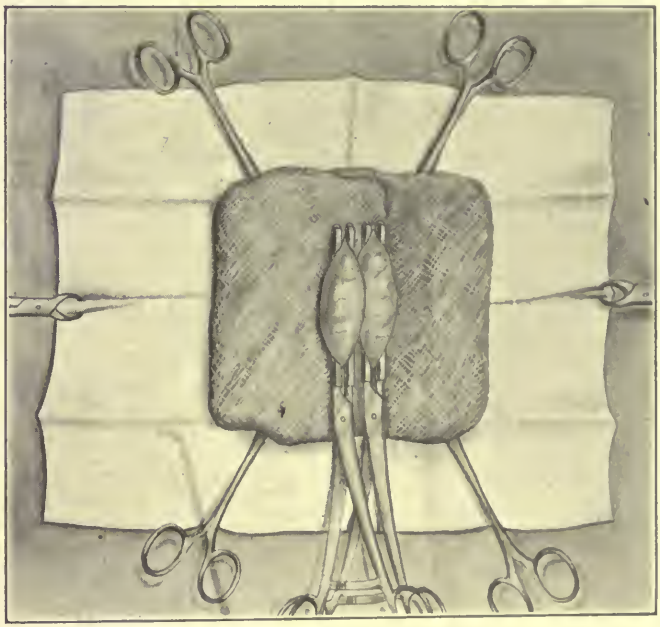


FIG. 878.— GASTRO-ENTEROSTOMY.

The clamps hold the viscera in position. The operative field is isolated by means of gauze pads.

is passed through the wall of the jejunum from the mucous to the serous surface at the left end of the incision, and then from the serous to the mucous surface of the stomach at a corresponding point; the knot, when tied, lies on the mucous membrane of the gut. The needle is now passed back and forth from the mucosa of the jejunum to that of the stomach, transfixing all the coats of the two viscera in its passage. When the suture has been completed along the posterior margin of the opening, it is returned along the anterior margin, with-

out interruption until the original end of the suture is reached, when a triple knot is tied and the ends of the suture are cut short.

The clamps are now loosened, but left *in situ* in order to prevent the viscera from slipping back into the abdomen, and the original serous suture is now completed. In doing this, the many vessels along the greater curvature of the stomach should be avoided; if one of these is punctured it must be doubly deligated at once. When this suture

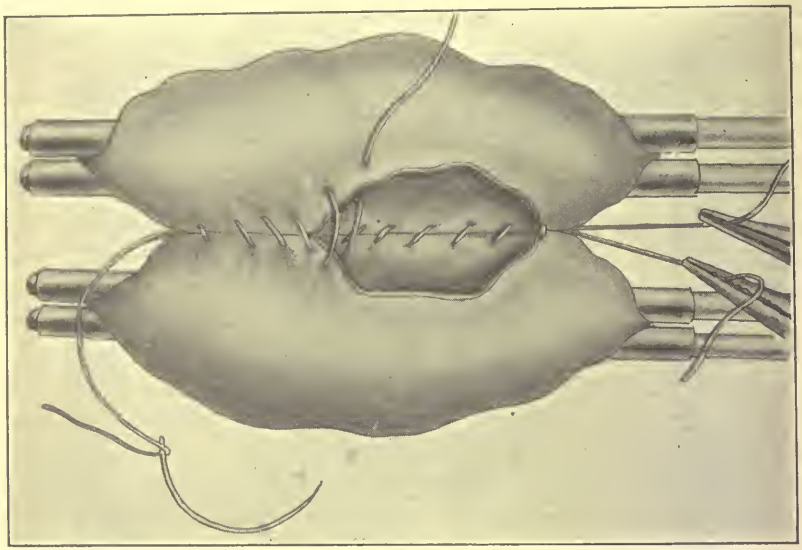


FIG. 879.— GASTRO-ENTEROSTOMY.

The inner suture nearing completion.

has been carried around to its point of origin, it is tied to the end of the first suture left long. The suture lines are now complete; the inner one embraces all the coats and is hemostatic; the outer approximates a wide surface of the serous coverings. A strip of gauze, it will be remembered, was placed between the two clamps. This is now utilized to rotate into view the entire line of anastomosis. If this appears defective in any part, it may be reinforced by a Lembert suture. The gauze strip is now removed and the transverse colon and stomach, which were replaced in the abdomen before the anastomosis was commenced, are now redelivered. The surgeon's left hand grasps the middle of the transverse colon and the lower part of the stomach and draws them gently away from the posterior wall of the abdomen, while

the right hand adjusts the portions of viscera at the anastomosis in the opening in the transverse mesocolon. A stitch now fastens the edge of this opening to the jejunum, first on one side and then on the other of the anastomosis. These stitches hold the transverse mesocolon in contact with the jejunum and prevent constriction in this situation.

FIG. 880.— GASTRO-ENTEROSTOMY.

Shows position of opening when the anastomosis is completed.

The stomach, colon and omentum are now replaced and the abdomen is closed.

ANTERIOR GASTRO-ENTEROSTOMY.— If for any reason *anterior gastro-enterostomy* should have to be performed, the method described in connection with the posterior operation may be employed. The only difference is that a point in the jejunum 15 to 18 inches from the flexure is chosen for the site of anastomosis; this avoids compressing the

transverse colon by the oral loop of jejunum. The method of anastomosis is very similar to the posterior method, but the stomach clamp is applied obliquely in such a direction that the adventitious stoma runs from above downward and to the left. In order to avoid kinking of the gut at the upper end, the jejunum is fixed to the stomach well beyond the opening. In the anterior operation entero-anastomosis is an advantage.

In cases in which the posterior operation is impracticable the anterior operation by Roux's method is preferable. In *Roux's operation*

FIG. 881.—ROUX'S OPERATION COMPLETED.

the jejunum is completely sectioned about 6 to 8 inches from the flexure; the aboral cut end is implanted into the stomach by end to side anastomosis and the oral end is implanted into the side of the aboral, about 3 inches from its union with the stomach. The anastomosis is preferably made between the posterior surface of the stomach and the aboral jejunum, but may be made with the anterior surface. The method is in many respects an ideal one — it simulates closely the normal conditions, the bile and pancreatic juice being introduced about 3 inches from the adventitious stoma, exactly as in the normal duodenum. The operation is not followed by regurgitant vomiting. The disad-

vantage of the method lies in its lengthy execution. In Roux's operation the stomach wall is clamped obliquely, so that the tip of the instrument corresponds to the lowest point on the greater curvature, while its heel grasps the stomach close to the lesser curvature near the cardia. An ample loop of jejunum — at least 8 or 9 inches — is utilized for the purpose. At this point the gut is clamped and divided 2 inches distal to it. The upper cut end of the jejunum is now united to the side of the lower part; the union is effected in the ordinary manner by suture. The distal cut end is united to the stomach.

The complications after gastro-enterostomy relate especially to *hemorrhage, regurgitant vomiting,* and *internal hernia.* Of these, regurgitant vomiting deserves special mention. Many theories have been advanced to explain its occurrence. There are four varieties of misdirected current after gastro-enterostomy.

1. Regurgitation of duodenal contents through the pylorus.
2. Escape of fluids from the stomach into the oral loop.
3. Escape of fluids from the oral loop into the stomach.
4. Regurgitation of the contents of the oral loop into the stomach.

Any of these establishes the *circulus visciosus.* Of the four, the third is generally considered the most frequent and the most grave.

Chlumsky,[18] in discussing the *causes of regurgitant* vomiting, suggests the following:

1. Formation of a spur. This is certainly the most frequent. When the anastomosis is effected, the two limbs of the loop hang downwards, and a sharp kink, with spur formation, results at the point of junction.

2. The jejunal displacement may cause a kink at the duodenojejunal junction, and thereby produce an acute obstruction of the duodenum.

3. The mucous membrane of the stomach may form large pouting valves which obstruct the afferent opening.

4. Closure of the anastomotic opening, if the mucous stitch is improperly applied.

If regurgitant vomiting persists, an entero-anastomosis, which drains the oral loop and the stomach, must be made (Moynihan[1]).

The development of *jejunal and gastrojejunal ulcer* sequential to gastro-enterostomy is a complication which dims not a little the brilliant results immediately following the operation. The subject is fully discussed by Van Roojen.[19] Moynihan[1] and his associates have collected 100 cases.

It seems not unlikely that the occurrence of a jejunal ulcer is due to the persistence of the factor which caused the chronic ulcer for the relief of which the gastro-enterostomy was performed. In the light of experience, the influence of lues in this connection must not be disregarded. The *operative remedy* lies in resection of the ulcer and the formation of a new gastrojejunal anastomosis — a difficult problem.

Operations for Chronic Gastric Ulcer.— The operation of *gastroduodenostomy* is usually performed by the method of Kocher[20] or Finney.[21]

Kocher[22] describes his operation as follows: "The most suitable incision is one similar to that which we recommend for exposing the gall-bladder, viz., an oblique incision two finger breadths below and parallel to the right costal margin, beginning at the middle line. After dividing the skin and fascia, the rectus muscle is cut through as far as the broad abdominal muscles. The posterior layer of the rectal sheath, the fascia transversalis, and the peritoneum are divided. In muscular subjects the transversalis muscle is split parallel to its fibers, which are then firmly drawn apart. The liver is drawn upwards, the stomach to the left, and the transverse colon and the descending limb of the hepatic flexure downwards. The duodenum is then brought into view, and its outer border is clearly defined. A pad of gauze is placed against the under surface of the liver, and the latter drawn well upwards with a suitable retractor. Gauze compresses are also employed to push aside the stomach and colon.

"The delicate layer of parietal peritoneum covering the kidney is divided vertically and 1 to 1½ inches external to the second part of the duodenum, and the incision is then continued vertically downwards through the upper layer of the transverse mesocolon (which is held on the stretch) as far as the larger branches of the vessels. The fingers are then introduced behind the left edge of the incision through the peritoneum, and the duodenum is separated from the vertebral column, the vena cava and the aorta, until it can be brought forward and pressed against the pyloric portion of the stomach, which, in its turn, is compressed against the left edge of the wound in the abdominal wall, so as to shut off the general cavity of the stomach and prevent escape of its contents. Both stomach and duodenum are now compressed above and below by the fingers of an assistant, and the lateral anastomosis is effected in the usual manner by two rows of sutures."

Moynihan[1] quotes Finney's description of his method as follows:

"Divide the adhesions binding the pylorus to the neighboring struc-

tures; also free as thoroughly as possible the pyloric end of the stomach and first portion of the duodenum. Upon the thoroughness with which the pylorus, lower end of the stomach, and upper end of the duodenum are freed depends, in large measure, the success of the operation and the ease and rapidity of its performance. A suture, to be used as a retractor, is taken in the upper wall of the plyorus, which is then retracted upwards. A second suture is then inserted into the anterior wall of the stomach, and a third into the anterior wall of the duodenum, at equidistant points — about 12 cm. — from the suture just described in the pylorus. These second sutures mark the lower ends of the gastric and duodenal incisions respectively. They should be placed as low as possible, in order that the new pylorus may be amply large. Traction is then made upwards upon the pyloric suture, and downwards, in the same plane, on the gastric and duodenal sutures. This keeps the stomach and duodenal walls taut, and allows the placing of the sutures with greater facility than if the walls remained lax. The peritoneal surfaces of the duodenum and stomach, along its greater curvature, are then sutured together as far as possible. For this row I would recommend the use of the continuous suture, as it is more easily and quickly applied and can be reinforced after the duodenum and stomach have been incised. After the posterior line of sutures has been placed, an anterior row of mattress sutures is taken, which are tied, but left long. These sutures, after they have been placed are retracted vertically in either direction from the middle of the portion included in the row of sutures. Then, after all the stitches have been placed and retracted, the incision is made in the shape of a horseshoe. The sutures should be placed far enough apart to give ample room for the incision. The gastric arm of the incision is made through the stomach wall just inside the lowest point of the line of sutures, and is carried up to and through the pylorus and around into the duodenum, down to the corresponding point on the duodenal side. Hemorrhage is then stopped. It is well to trim off with scissors redundant edges of mucous membrane, in order to prevent the formation of a valve-like fold of mucous membrane at the new pylorus. A continuous catgut suture is now taken through and through all the coats of the intestine on the posterior side of the incision. This reinforces the posterior line of sutures, secures better approximation of the cut edges of the mucous membrane, and prevents the reunion of the divided intestinal walls. The anterior sutures are then straightened out and tied, and the operation is complete, unless one wishes to

reinforce the mattress sutures with a few Lembert stitches. This procedure, as is readily seen, gives the minimum of exposure of the infected surface. All the stitches are placed and the posterior row tied, before the bowel is opened, and it remains open just long enough to control the hemorrhage. The size of the newly formed pyloric open-

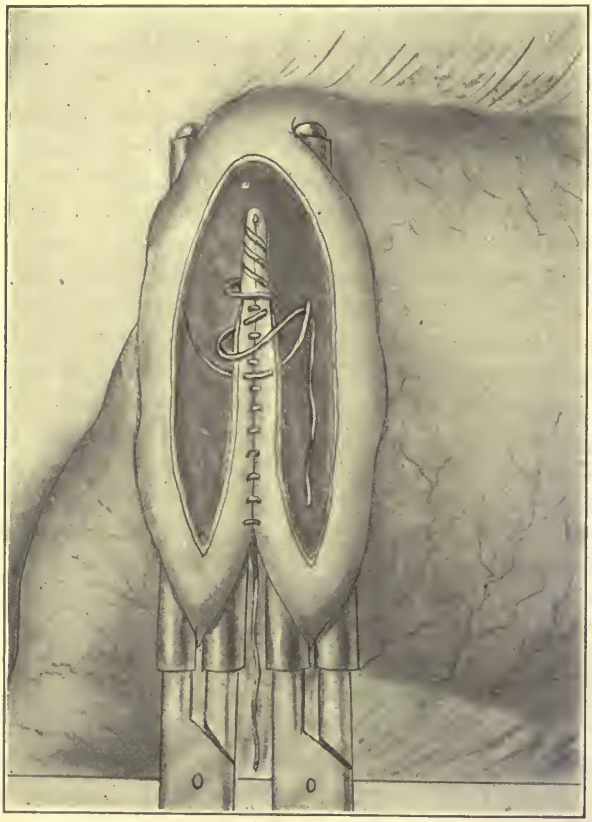


FIG. 882.—FINNEY'S OPERATION.

The first line of suture is completed; the inner is commenced.

ing is limited in this operation only by the mobility of the stomach and duodenum and the judgment of the operator.

"In all of my cases the incision has been about 12 cm. in length, and could have been made longer had I chosen to make it so. Unless the stomach is very much dilated or has descended to an unusual ex-

tent, the lower limit of the new outlet is at or near the level of its most dependent portion."

Excision of Gastric Ulcer.— In connection with the surgical treatment of ulcers of the stomach, the following general procedure may be regarded as established:

1. In cases of cicatricial obstruction, the result of healed ulcer

FIG. 883.— SECTIONAL RESECTION OF STOMACH.

The omenta are ligated. The dotted lines correspond to the sections to be made.

at, or near, the pylorus, posterior gastro-enterostomy affords permanent relief.

2. Ulcers located in parts of the stomach other than the pyloric antrum are best excised.

3. Excision of an ulcer upon the lesser curvature may be practiced if the lesion is small, otherwise sectional resection of the stomach is indicated (Stewart[28]).

4. An ulcer on the posterior wall of the stomach may be reached by the transgastric route, but is best treated by sectional resection.

5. Large ulcers lying near the pylorus are best treated by partial gastrectomy, occlusion of the duodenum, and gastro-enterostomy.

It will be seen that, except for small ulcers of the lesser curvature, resection of large or small sectors of the stomach is the method of choice. The operation is performed as follows:

The two curvatures of the stomach are freed from the omenta as widely as is necessary by ligature of the coronary and gastric epiploic vessels above and below. The doomed sector of the stomach is thus denuded of its blood supply. At each end of this sector two pairs of

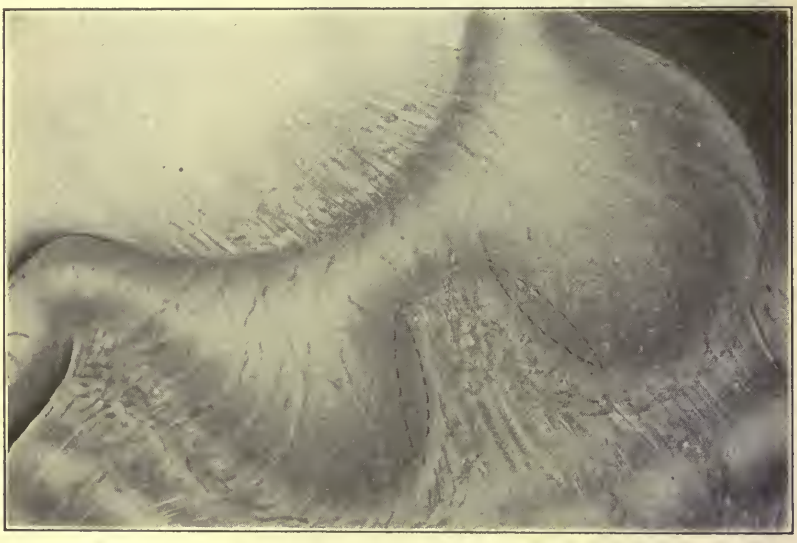


FIG. 884.— HOURGLASS STOMACH.

The dotted lines indicate where the openings for gastrogastrostomy are made.

clamps are placed; between each two the stomach is divided and the diseased section is removed, the divided ends being united by end to end anastomosis.

HOURGLASS STOMACH.— Hourglass stomach is best treated by *gastrogastrostomy*. The openings in the pouches correspond to the dotted lines shown in Fig. 884, or the constricted portion of the stomach may be resected in the manner just described.

RESECTION OF THE STOMACH FOR CANCER.— Resection of the stomach for cancer must take into account the lymphatic system of this organ (Cuneo,[24] Lengemann,[25] Jamieson and Dobson[26]). Observations

in this connection indicate that lymphatic drainage occurs toward the pylorus and lesser curvature (Fig. 885).

The operation of *partial gastrectomy* is performed as follows:

An upper median celiotomy is made; this is followed by examination of the lesion, which decides the extent of the operative procedure. As stated elsewhere, neither adhesions nor the involvement of lymphatic glands necessarily precludes removal of the stomach.

The arteries which supply the stomach are secured first. The first of these, the pylorie artery, is given off elose to the upper border of the pylorus by the hepatic artery. It may arise separately, or from a

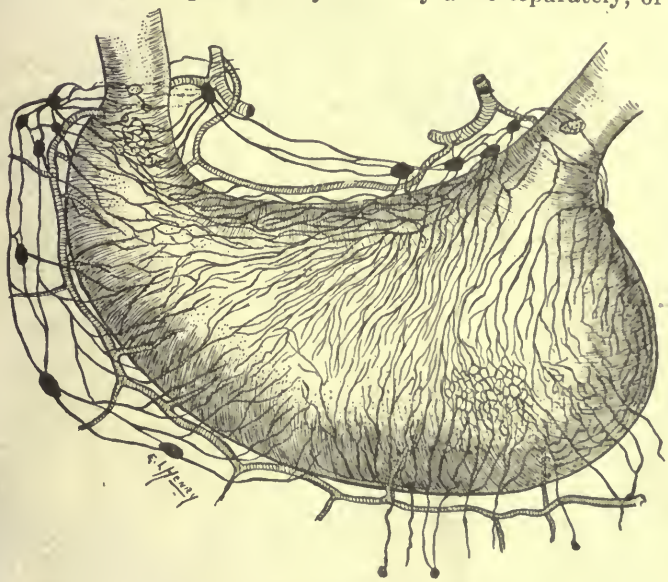


FIG. 885.— LYMPHATICS OF STOMACH.

common trunk together with the gastroduodenal artery; if separately, it is doubly deligated and divided as far from the stomach as is possible. From this point the peritoneum is sectioned above the pylorus and the first part of the duodenum in such a manner as to make it feasible to strip all the glands upward toward the stomach. In this step the common duct must be avoided. The left forefinger is then passed from behind the duodenum from above downwards, separating its first part from the pancreas, and is made to project at the lower border of the bowel to the right of the subpyloric group of glands. As the finger is withdrawn, two clamps are passed along its tract,

both embracing the duodenum. The latter is then sectioned about 1 inch from the pylorus, close to the aboral clamp. At once the mucosa of the oral stump is cauterized. At this point, the divided end of the duodenum is turned to the left, exposing the right gastric epiploic artery as it is given off from the gastroduodenal; the former is now tied. As the aneurism needle surrounds the vessel, the colon and omentum are turned upward, displaying the middle colic artery, the integrity of

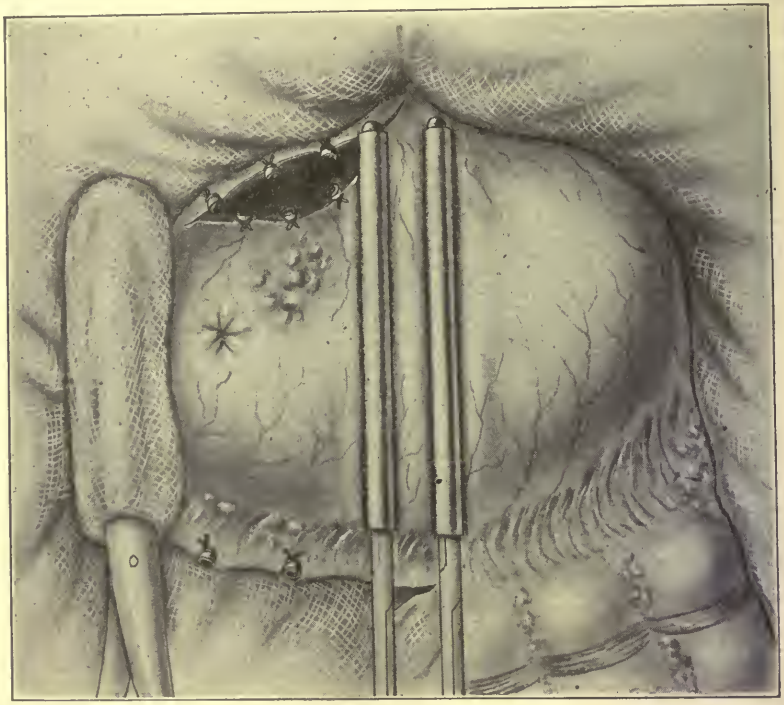


FIG. 886.—PARTIAL GASTRECTOMY.

which must be preserved. The aboral end of the duodenum is now closed by means of a double row of sutures. The next step consists in division of the gastrocolic omentum along the greater curvature of the stomach. The concavity made by the second portion of the duodenum is cleared of all fat and glandular elements. It is important that this subpyloric group of glands, together with those lying in the gastrocolic omentum, be included in the parts to be removed. As the stomach is lifted from the pancreas, the retropyloric glands may

be seen, and adhesions to the pancreas discovered. The latter must be carefully separated. A series of ligatures is passed through the gastrocolic omentum from right to left, care being taken not to wound nor include the gastrocolic artery. Occlusion of this vessel is followed by gangrene of the colon. This serial ligature is continued until the middle of the greater curvature of the stomach is reached, and at this point the gastro-epiploic vessels are ligatured and divided. At this stage of the operation the stomach is readily turned over to the left, the posterior surface being now to the front, thus freely exposing the entire anterior surface of the pancreas. Gentle traction on the stomach makes visible the coronary artery as it arises from the celiac axis; it is surrounded by a double ligature and divided. The upper coronary group of glands is stripped from the vessel before it is ligatured. Section of this artery mobilizes the stomach, which may now be readily turned still farther to the left. While it is held in this position the anastomosis with the jejunum is made. If the adventitious stoma is made after the stomach has been divided, the technical difficulties of this step of the procedure are much increased. The steps of the anastomosis are similar to those described in connection with gastrojejunostomy. The stomach itself is now divided between two pairs of large curved clamps, their convexity turned toward the pylorus. When the clamps are secure the stomach is cut across about three fourths of an inch aboral to the gastro-enterostomy opening. The serous and muscular coats are first divided and pushed back from the mucosa, which is then sectioned as near as possible to the proximal clamp. The opening in the stomach is closed in the usual manner. Glands are now sought for in the course of the hepatic artery at the upper border of the pancreas and removed. The omentum and the transverse colon are turned upwards and the opening in the posterior mesocolon stitched to the gastrojejunal suture line in the usual manner. Drainage is not employed unless the pancreas or the common bile duct has been injured.

Gastrostomy.— Gastrostomy consists in making an opening directly into the stomach for the purpose of administering nourishment in cases of obstruction of the esophagus or cardiac end of the stomach. Many methods have been devised for the purpose. Of these, two, which encompass the principles of all, are described here. In Kader's[27] method a vertical incision about 3 inches in length is made through the outer border of the left rectus muscle. The stomach is delivered into the wound and a portion of its anterior wall folded

inward by interrupted sutures passed above and below a tube introduced through a small incision The tube is fastened to the edge of the incision in the stomach by a single catgut stitch. Two vertical parallel folds of the stomach are next raised up on each side of the tube by four or six sutures. When these sutures, which include only the serosa and muscularis, are tied and cut, another series is introduced,

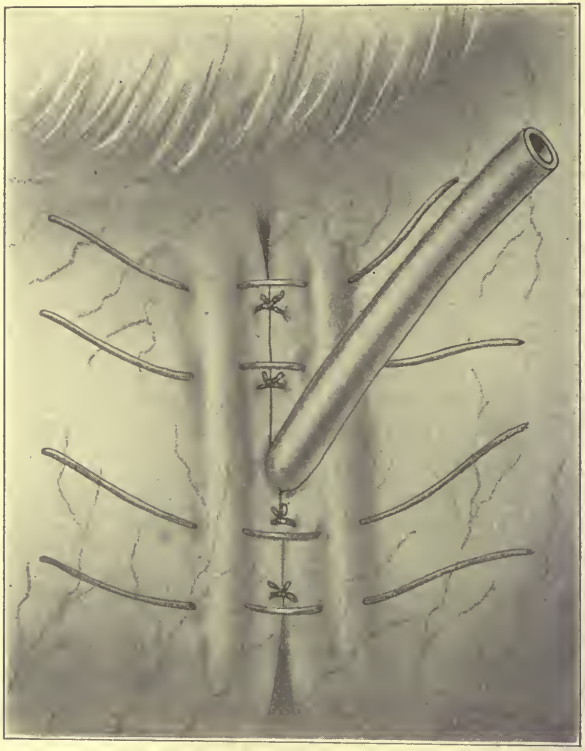


FIG. 887.— KADER'S GASTROSTOMY.
Second (infolding) row of sutures in place, but not tied.

which picks up the stomach wall on each side of the original line of stitches (Fig. 887). On tying this second row, the first is buried. The stomach is then sutured to the anterior abdominal wall. Thus a valvular stoma is fashioned.

The method of Witzel[28] is performed as follows:

The stomach is brought into the wound and opened by a small incision, into which a tube is fastened in the manner stated above.

The tube, when secured, is laid against the anterior wall of the stomach and buried in a sort of gutter formed by raising up a fold on each side of the tube by means of a continuous suture (Fig. 888). About 2 inches of the tube is thus covered and made to lie in a canal in the stomach wall. The stomach is then fastened to the anterior abdominal wall through which the tube is made to protrude.

JEJUNOSTOMY.— Jejunostomy is practiced as a palliative measure in cases of lesions of the stomach which do not permit of gastro-enter-

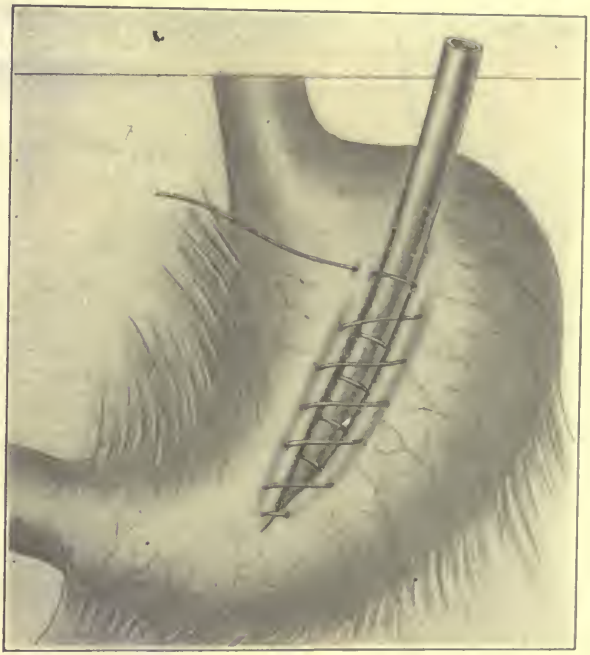


FIG. 888.— WITZEL'S GASTROSTOMY.

ostomy. It has a limited field of usefulness (Maydl[29]). The abdomen is opened through the left rectus muscle and the jejunum delivered and cut across. The oral end is implanted into the side of the jejunum farther down, and the aboral end is sutured into the abdominal wound. The latter is used as a feeding stoma. For additional methods see Moynihan,[30] and Robson.[31]

Intestinal Suture.— Of the numerous methods devised for the purpose of repairing or anastomising the gut, the following are used by most surgeons of the day and are sufficient for all practical purposes.

The *Lembert suture* is the simplest of all intestinal stitches. Its principle is the same for the continuous as for the interrupted suture. The needle, threaded with a fine material (silk, linen or gut), is introduced transversely to the wound. It transfixes on each side all the coats except the mucosa, and is introduced about ¼ of an inch, or

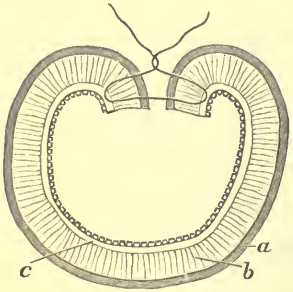


FIG. 889.— LEMBERT SUTURE.

a, Serous coat; *b*, muscular coat; *c*, submucous fibrous coat.

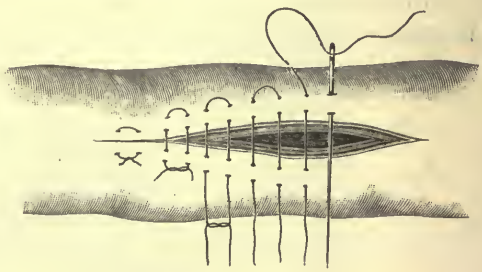


FIG. 890.— THE HALSTED SUTURE.

Closure of a longitudinal incision.

less, from, and emerges about a line from the edges of the wound on one side, then passes across the wound to enter on the oppostie side at a point immediately beyond the opening, there to engage the wall of the gut in the same manner as on the opposite side (Fig. 889).

Halsted's, or the mattress, suture is in reality the equivalent of a double Lembert suture. The needle, having passed from one side of the wound to the other, is made to return so that the two ends of the suture lie upon the same side (Fig. 890). The stitch brings broad surfaces of peritoneum into apposition and is a very satisfactory one.

FIG. 891.— THE CUSHING SUTURE.

Cushing's suture is continuous and, as it is introduced parallel to the wound, accomplishes the same purpose as the Halsted (Fig. 891).

The *Connell suture* is used to repair wounds of the gut, to unite its ends after complete transverse section, or for the purpose of lateral anastomosis. The suture may be interrupted or continuous. At the

present time, the latter is mostly used. However, for the purpose of clearness, the method of making the repair with interrupted stitches is quoted as described by Connell.[32]

"In using the interrupted suture, the first stitch should be taken at the mesenteric attachment. This stitch is of the greatest importance on account of the separation of the serous covering of the bowel at this point, and great care should be exercised in securing a perfect approximation at this point. In order to do this in the most satisfactory manner the stitch should be introduced as follows: the needle is made to enter the bowel wall of one cut end from the lumen, per-

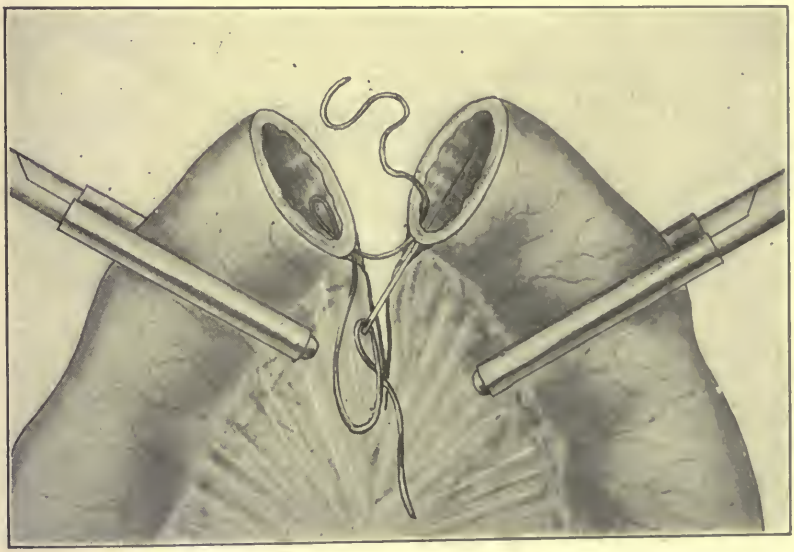


FIG. 892.— THE CONNELL SUTURE, *A*.

forate all coats, and pass through the serosa of one side of the triangular space formed by the separation of the serous coats; then on, over and through the serosa of the opposite cut end, at the same relative point, i. e., the side of the triangular space, then on through the wall into the lumen. This completes one half of the stitch, and is made with one movement of the needle.

"The needle is next reversed, and at a distance of about 3 mm. is made to repeat the steps in the opposite direction, i. e., entering the mucosa of the second cut end, passing through all coats of the bowel wall, including the serosa of the triangular space, and then through

the serosa of the triangular space of the first cut end, on through the wall, into its lumen, where the needle end and the free end of the suture are tied in a knot on the mucosa. This stitch, which acts similarly to a brad, absolutely secured a perfect serous approximation at this point, which is considered by all to be the most difficult portion of an enterorrhaphy.

" The remainder of the union is closed by stitches exactly similar to this first stitch. The cut ends are held in proper position by any convenient means, such as the Lee holder, the Allis tenaculum forceps, or suspending loops of thread; but the best method in my experience

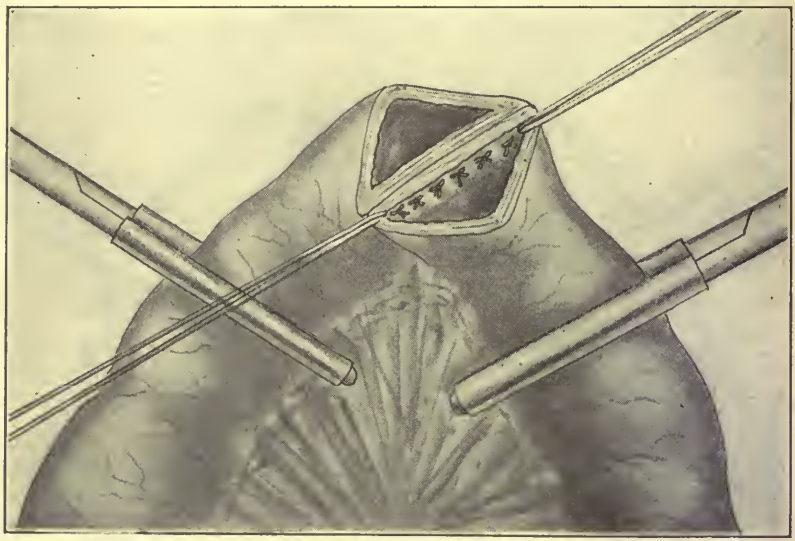


FIG. 892.— THE CONNELL SUTURE, *B*.

is the plan of introducing a stitch, such as the other stitches, at a conveniently distant point, and leaving the ends of this stitch long, to be held by an assistant while the intervening stitches are being inserted.

" This method has the advantage of consuming no additional time, for when the union is completed up to this stitch and the long ends are of no further use, they are cut away, leaving the stitch itself in place.

"With the ends thus held in proper position, the introduction of the stitches is a very simple matter — merely one passage of the needle through all coats of both cut ends, and then at a distance of about ⅛

of an inch the same process in the opposite direction, and, finally, the tying of the knot upon the mucosa at one side of the seam.

"This is repeated until all but a small portion of the union is completed. In order to place the last stitch and knot in exactly the same relative position as the first ones, we proceed in this manner:

"The needle enters the gut wall from the lumen, passes through all coats, and emerges from the serosa of one side. The needle is then turned upon itself and made to retrace its steps at about ⅛ of an inch distant, passing from the lumen through all coats, emerging from the

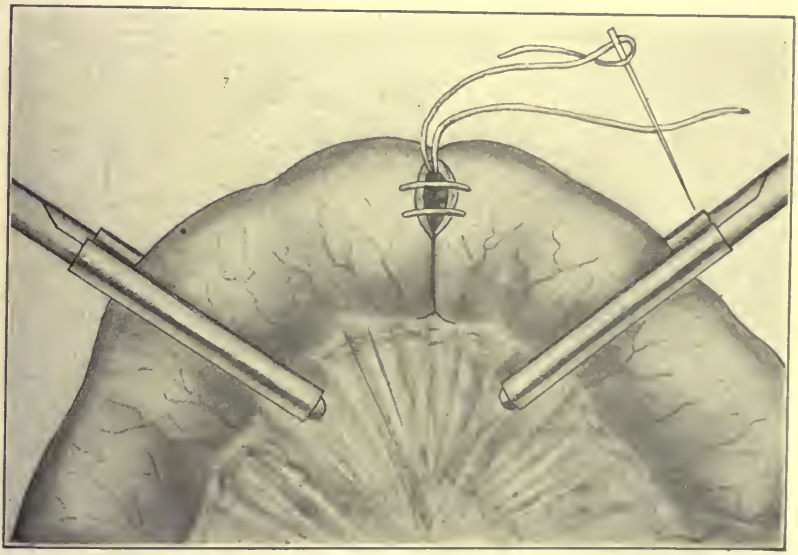


FIG. 892.— THE CONNELL SUTURE, *C*.

serosa, then over to the opposite side and emerging through the serosa, and, finally, ending in the lumen of the cut end from which it began.

"Now the two ends of the thread which are to make the knot are side by side, emerging from the mucosa into the lumen, and then emerging from the ununited part of the enterorrhaphy out of the body. The needle, in introducing this stitch, has passed through the cut ends in exactly the same order as it did when inserting the stitches in the earlier part of the operation when the cut ends were held in sero-serous apposition — i. e., mucosa, submucosa, muscularis, and serosa, then immediately on into the other cut end through the serosa, muscu-

laris, submusoca, and the mucosa, and then in this order repeated in the opposite direction ⅛ of an inch away.

"The needle and the free end of the suture now hang side by side from the mucosa, and in order that they may be tied in a firm knot, proceed as follows:

"At a point in the line of union, about opposite this last and still untied stitch, a threaded needle is inserted, eye first, between two of the previously inserted and tied stitches. The needle is passed between the apposed serous surfaces into the lumen.

"By passing the needle still farther onward it is made to present

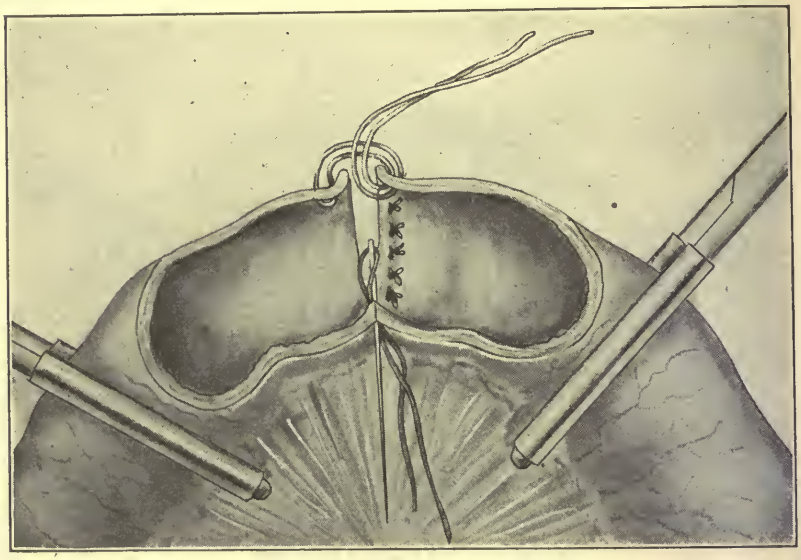


FIG. 892.— THE CONNELL SUTURE, *D*.

at the location of the last stitch, where the ends of the suture still protrude, and where the surfaces are not united. By slightly withdrawing the threaded needle a loop is formed with its thread; into this loop are placed the two free ends of the last stitch which is to be tied. By withdrawing the needle and in its loop the stitch ends, these ends will be made to present upon the peritoneal aspect of the bowel on the opposite side, between two of the previously inserted and tied stitches, i. e., at the point where the threaded needle was inserted. Slight traction upon these ends will cause the remaining portion of the line of union to become inverted, and seroserous approximation

will obtain entirely around the site of suture. Upon greater traction the bowel will become flattened, bringing the mucous membrane upon which the last knot is to be located into intimate relationship with the line of suture at the point where the free ends protrude. The knot is tied with the bowel in this flattened position, thus avoiding the occurrence of any slack. While still retaining the tension, the ends of the knot are cut off short, so preventing any long free ends in the lumen. Upon allowing the bowel to assume its normal contour — that of a cylinder — the knot will slip between the already tied stitches into the

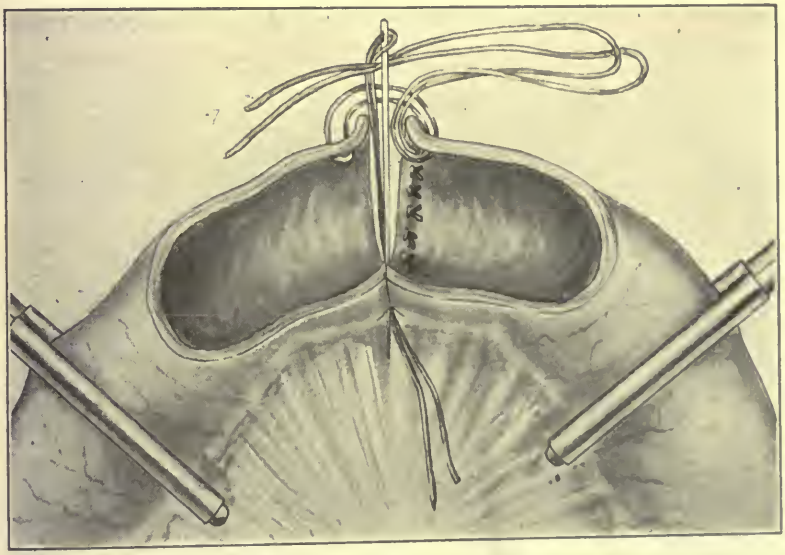


FIG. 892.— THE CONNELL SUTURE, *E*.

lumen, and, as it is attached to the mucosa of the opposite pole of the diameter of the gut, it goes with that portion of the bowel wall."

Enterotomy and Enterostomy.— *Enterotomy* is occasionally performed for the removal of an intestinal obstruction or foreign body. As a rule, the opening is made to give egress to the accumulation of the intestinal contents (which is very toxic) above the point of obstruction.

A point about 10 inches above the site of the obstruction is chosen for the opening. A loop of gut is delivered through the celiotomy wound, emptied by stripping, and clamped by the fingers of an assistant. A longitudinal incision, about 1 inch in length, is then made in

the gut opposite the mesentery. The edges of this incision are seized with Allis' forceps. The aboral portion of the gut, i. e., that between the obstruction and the incision, is now emptied. A glass tube from 6 to 8 inches in length, to the outer end of which a long rubber tube is attached, is introduced into the opening in the gut and gently pushed upward from 3 to 4 inches. The forceps are removed and the gut is gradually drawn more and more over the glass tube, emptying the contents of the former. With care 8 to 10 feet of intestine can be telescoped in this way (Fig. 893). Finally, the glass tube is slowly withdrawn and the opening in the bowel closed with a Lembert suture.

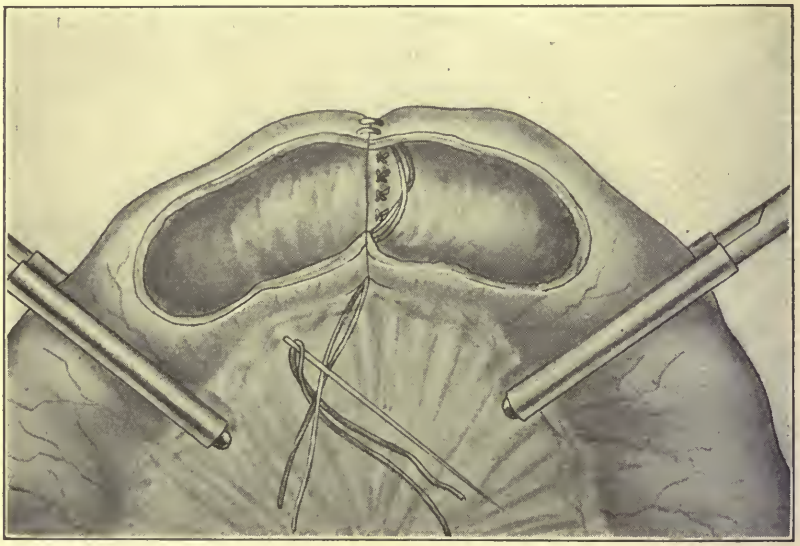


FIG. 892.— THE CONNELL SUTURE, *F*.

Enterostomy consists in fastening the gut to the abdominal wall and opening it for the purpose of allowing the escape of its contents. Two forms of openings are made. In the one, the small intestine is opened temporarily for drainage, i. e., a *fecal fistula* is made. In the other, the large intestine is opened for permanent drainage, i. e., an *artificial anus* is established.

Temporary drainage of the intestine is provided in cases of acute intestinal obstruction as a measure of relief in menacing cases. The *operation* is simple. The incision is made through the lower right rectus muscle, and a low loop of the ileum is fixed to the parietal

wound. Two sutures, one at each end, are introduced first; these pass through all the layers of the abdominal wall on one side, then pick up a broad surface of visceral serosa and transfix the abdominal wound on the opposite side. The edges of the parietal peritoneum are now sutured to the gut presenting through the celiotomy wound. When this suture is completed the two fixation sutures are tied. Opening the gut is postponed for several hours, when a purse string suture is introduced into the area of gut presenting. Within this the gut is opened and a Paul tube, or similar device, is introduced through the opening and held in place with the purse string suture.

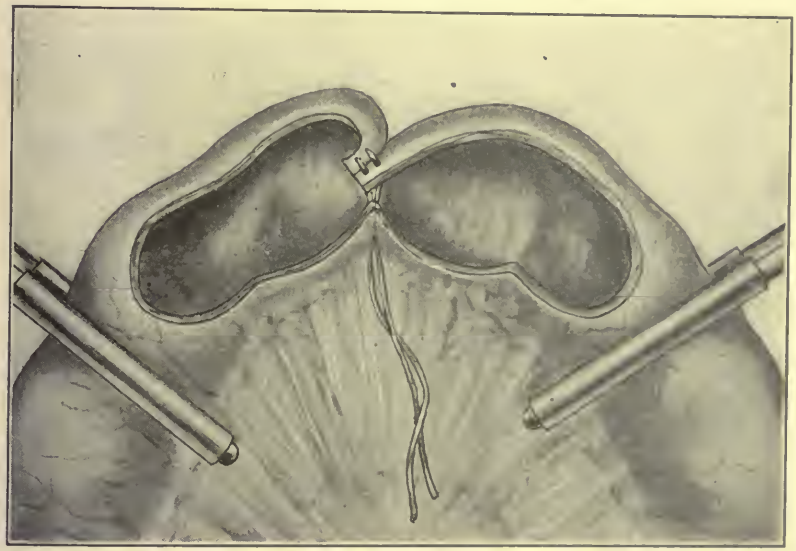


FIG. 892.— THE CONNELL SUTURE, *G.*

COLOSTOMY.— Colostomy may be performed through a muscle splitting celiotomy wound or through what seems to be the *dernier cri* in this sort of surgery, a straight celiotomy incision placed below and about 1 inch to the left of the umbilicus. Sistrunk,[83] who devised the method, describes the subsequent steps of the operation as follows:

"The colostomy is made in a loop of the sigmoid flexure; this portion of the bowel is chosen because of its extreme mobility and the length of its mesentery. After the loop has been lifted out of the abdominal cavity, an incision from 1½ to 2 inches in length is made through its mesentery, parallel to the direction of the blood vessels,

and extending upward nearly to the bowel. A second incision, about 1 inch long, is then made across the end of the first incision quite near the mesenteric attachment of the bowel. Two strips of gauze are placed in the upper ends of this T incision, and when these are pulled apart a goodsized opening through the mesentery is created. Two sides of the entire abdominal wall near the center of the abdominal incision are sutured through this opening in the mesentery. The remaining portions of the incision are then closed; a small bite of the bowel is included in two of the sutures to prevent the possibility of herniation of a loop of small intestine. When the gauze has been

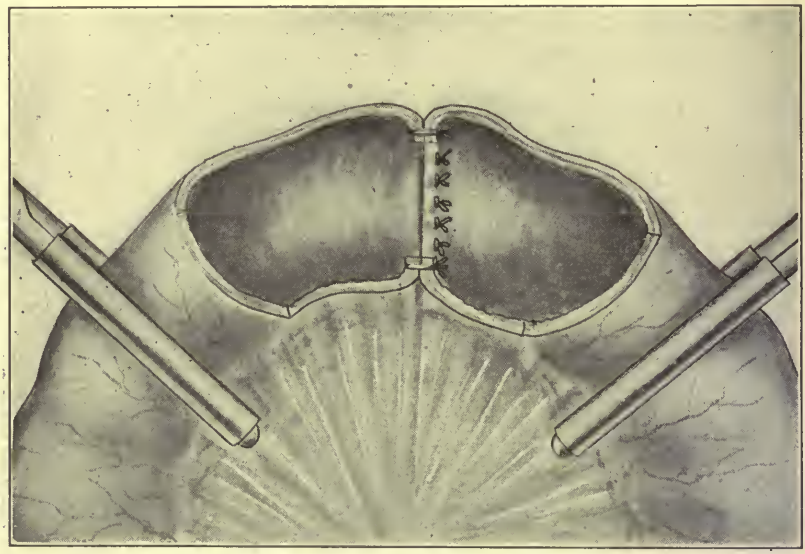


FIG. 892.— THE CONNELL SUTURE, *H*.

removed a glass rod or rubber tube is placed through the opening in the mesentery between skin and bowel to serve later as a guide in cutting across the bowel.

"If necessary for gas distention, a small opening may be made in the bowel any time after twenty-four to forty-eight hours, and in from five to six days the bowel is cut across completely with the cautery. As a rule, however, the patients pass gases very readily through the knuckle which has been brought up, and it is unnecessary to disturb it before 5 or 6 days. After the loop has been cut across it will be found that the two ends of bowel are separated by the entire abdominal

wall for a distance of from 1 to 1½ inches and that the cut ends of bowel protrude for a distance of an inch or two above the skin. These may later be cut shorter if it is thought necessary."

Entero-Anastomosis, Lateral Anastomosis, Short Circuiting.— In lateral anastomosis, or short circuiting, of the gut a communication is established between the bowel above and below the site of a lesion.

The important indications for the operation are as follows:

1. Intestinal obstruction due to growths.
2. Simple stricture due to a cicatricial contraction following strangulated hernia, etc.
3. Tuberculous disease of the intestine with extensive and inseparable adhesions, with the matting together of a large area of the intestine.

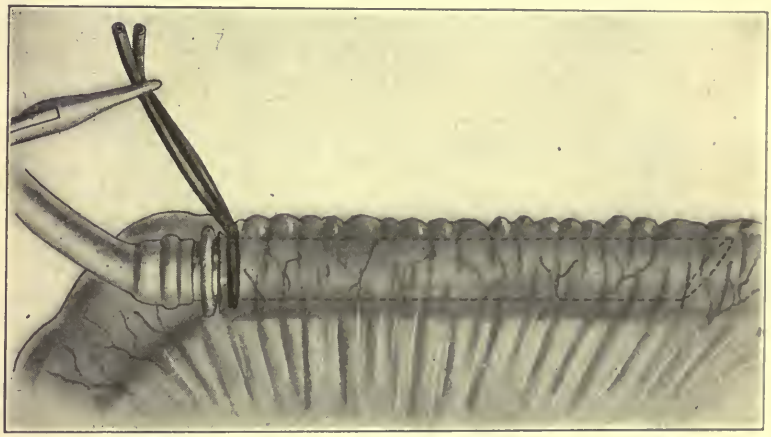


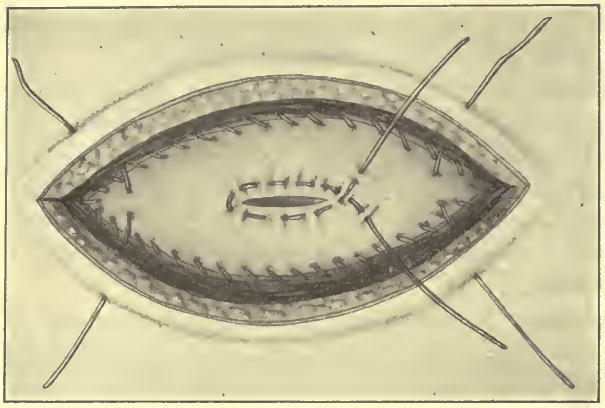
FIG. 893.— EMPTYING THE GUT THROUGH AN ENTEROSTOMY OPENING.

4. Multiple adhesions due to sclerosing peritonitis of various origin.
5. Intestinal exclusion.

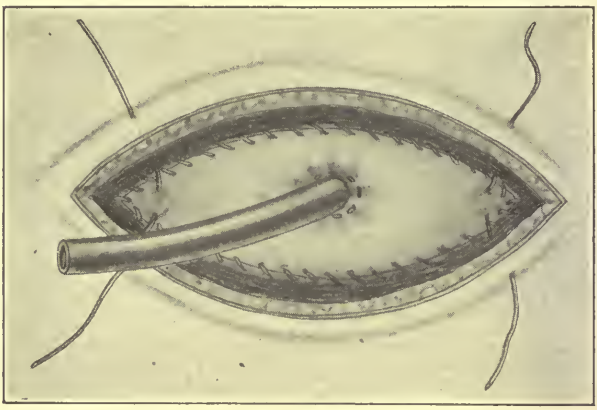
As far as the means of effecting the anastomosis is concerned, none, other than simple suture, is considered at this writing. The technic of the procedure does not differ in any regard from that already described in connection with posterior gastro-enterostomy (p. 1919).

ENTERECTOMY.— Enterectomy is performed for relief of the following conditions:

1. New growths.
2. Strictures of the gut from various causes.



A



B

FIG. 894.— TEMPORARY ENTEROSTOMY.
A, Gut sutured to wound. *B*, Purse string tied to tube.

3. Gangrene of the gut.
4. Irreducible intussusception.
5. Multiple intestinal perforation.
6. Extensive lacerations of the intestines.
7. Certain classes of fecal fistulae.
8. Growths of the mesentery which endanger the vitality of the gut.

In resection of the small intestine the doomed sector is carefully isolated and drawn well out of the abdomen. The limits of the gut

to be resected having been determined, the intestinal clamps are applied. Four pairs are necessary — two pairs at each point. The clamps are applied at the angle shown in Fig. 896. The gut is now divided at each end of the sector to be excised, and the cut ends cleansed, after which the ends of the intestine are temporarily wrapped in wet gauze, so that none of the contents comes in contact with the

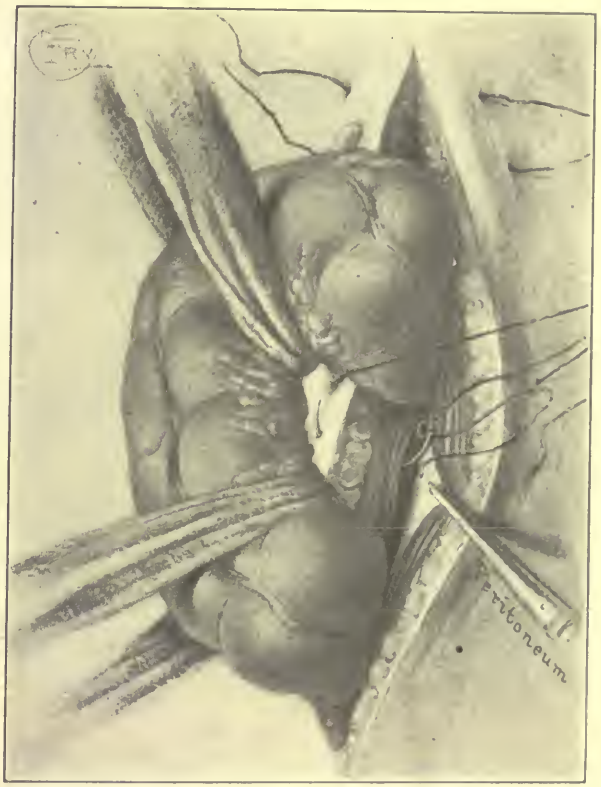


Fig. 895.— Transrectus Colostomy (Sistrunk).

operative field. The incision is carried onwards obliquely into the mesentery, or the apex of the wedge of mesentery to be removed is isolated in a broad clip, which is firmly closed and allowed to remain for a few moments. When it is removed a deep groove will be seen, the vessels having been obliterated by the pressure of the forceps. The groove is now ligated with chromic gut (00). A clamp is then applied 1 inch distal to the ligature and the mesentery between them

is divided; the affected loop is now removed. The excised portion includes the lesion, a length of healthy bowel on either side, and a wedge of mesentery containing the lymphatics and glands connected with the affected gut. The conditions at this stage of the procedure are shown in Fig. 896. The subsequent steps of the operation are similar to those described in connection with intestinal suture by the

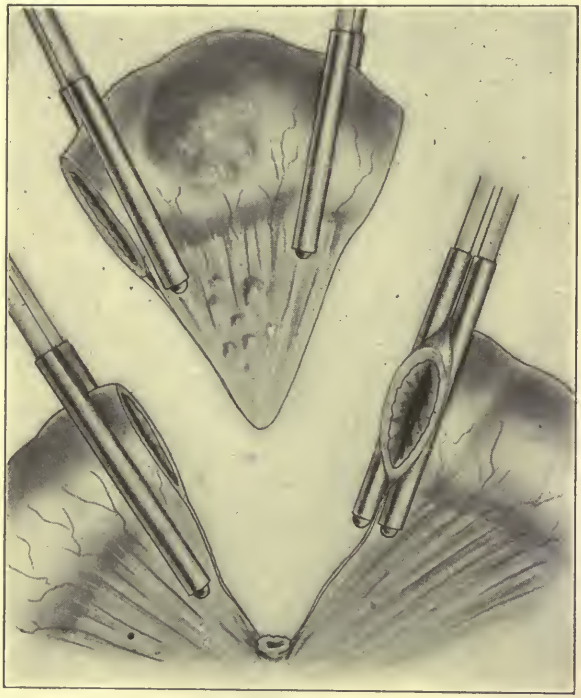


FIG. 896.— ENTERECTOMY.

Connell method (p. 1937). The question of the length of gut that it is permissible to remove is discussed at great length by Flint.[34]

In *resecting the colon* the choice between the two methods of anastomosis — side to side or end to end — has exercised the minds of surgeons for years. Moynihan[1] summarizes the situation as follows:

"There are advantages in both. If the peritoneal investments are complete in both ends to be united, then end to end anastomosis seems to me the best method. It is, however, far more difficult to do with accuracy, neatness and precision; if done in a slipshod manner, disaster is bound to follow, leakage will occur from the suture line, and

a general peritoneal infection or a localized abscess will be the result. If the serous covering is incomplete in one or other segment of the bowel, then I think it more prudent to close the two cut ends, and to approximate their serous surfaces in a side to side anastomosis, which should always be of ample size. Healing in such an attachment occurs readily, and the union is sound and firm within a few hours. It has been supposed, and the supposition receives undoubted support from experimental observation, that an end to end anastomosis allows a more

FIG. 897.— ILEOCOLIC RESECTION.

The cecum and ascending colon are stripped up.

easy onward progression of the intestinal contents. It may be so, but I have never seen any difficulty arise in my own cases when the anastomosis has been made laterally; and as W. J. Mayo[35] points out, the side to side anastomosis, if seen a few months after its formation, differs from the end to end very slightly in appearance — a small elbow alone marks the point at which the junction has been made."

Ileocolic resection (Friedrich[36]) illustrates so many of the problems, and the method of dealing with them, connected with intestinal surgery,

that the following description seems to be justified and may be regarded as applicable to all portions of the large and small intestine. The abdomen is opened by a transrectus incision 6 to 8 inches in length. The small intestine, with the exception of the last 8 inches of ileum, is pushed well over to the left and into the pelvis, and protected by hot moist pads; another large pad is placed above the hepatic flexure, holding aside the stomach and liver, and a third fills the iliac fossa below the cecum. This maneuver isolates and exposes the parts to be removed, i. e., the terminal 5 or 6 inches of the ileum, the cecum and appendix, the ascending colon and 3 or 4 inches of the transverse colon.

FIG. 898.— ILEOCOLIC LATERAL ANASTOMOSIS, FOLLOWING RESECTION OF THE CECUM AND ASCENDING COLON.

By dividing the posterior peritoneum at the outer side of the ascending colon, the latter is easily mobilized with the fingers. In stripping the colon from the posterior abdominal wall, the ureter must be avoided. At the upper end of the incision the third portion of the duodenum and a part of the second are exposed, the peritoneum being raised from the duodenum to where the superior mesentery artery crosses it. The stripping is continued at the upper end until the hepatic flexure is mobilized and the cecum is freed. The terminal part of the enteric mesentery on its lower surface will be divided for a short distance. The surgeon's hand is now passed behind the ascending colon up to the

third portion of the duodenum. The peritoneum is made tense by the fingers, and a small incision is made into it from in front. This opening is enlarged until the superior mesenteric artery with the ileocolic branch is exposed. The exact position of the ileocolic artery and of the right colic artery is now readily determined. By delivering the ascending colon out of the wound, the precise position of these vessels may be seen through the translucent peritoneum. The ileocolic artery is now doubly deligated and divided. If the right colic artery arises separately, it must be tied by itself. Two incisions are now carried through the peritoneum, the one extending downward to the ileum and the other upward and outward to the beginning of the transverse colon. In the former incision the superior mesenteric artery is ligated. The large branch of the middle colic, which anastomoses with the right colic, must be secured by ligature and divided from 2 to 3 inches from the gut. At this stage of the operation, the part to be removed, with a large triangular piece of mesentery and mesocolon containing the terminal part of the superior mesenteric artery, the right colic artery, and a part of the middle colic, with the lymphatics and glands lying along them, are now attached only to the bowel above and below. The gut at each end is divided between clamps and the mass is free. The division of the bowel and the subsequent anastomosis is made by the lateral ileocolic method; that is to say, the final step of the anastomosis is carried out in the manner described in connection with posterior gastro-enterostomy (p. 1919). The completed intestinal suturing is shown in Fig. 898. The raw surfaces are covered by approximating the edged of the stripped out edges of the posterior peritoneum.

Removal of the *hepatic flexure*, *transverse colon*, *splenic flexure*, and *descending flexure* should be followed by closure of the divided ends and lateral anastomosis. The absence of peritoneum from more or less of the surface of the large gut makes end to end anastomosis undesirable (though this is technically possible and has been frequently accomplished). A similar principle may be followed in connection with resection of the *sigmoid flexure*. However, in this situation the operation devised by Morrison,[37] perhaps with slight modification, is frequently employed. The technic of the procedure is described by the originator of the method as follows:

"1. The diseased bowel is excised.

"2. A glass bobbin with india rubber tube affixed is tied tightly into the upper end of the sigmoid flexure.

"3. The india rubber tube is passed down from above through the

lower cut end into the rectum, where it is aided in passing through the anus by the finger of an assistant. If there be any difficulty in effecting this, the assistant passes a stomach tube up from the anus into the belly. A piece of string is tied to the stomach tube and to the india rubber and all are drawn back through the anus.

"4. The tube is drawn upon until the ligature on the upper cut end of the bowel is inside of the lower cut end of the bowel.

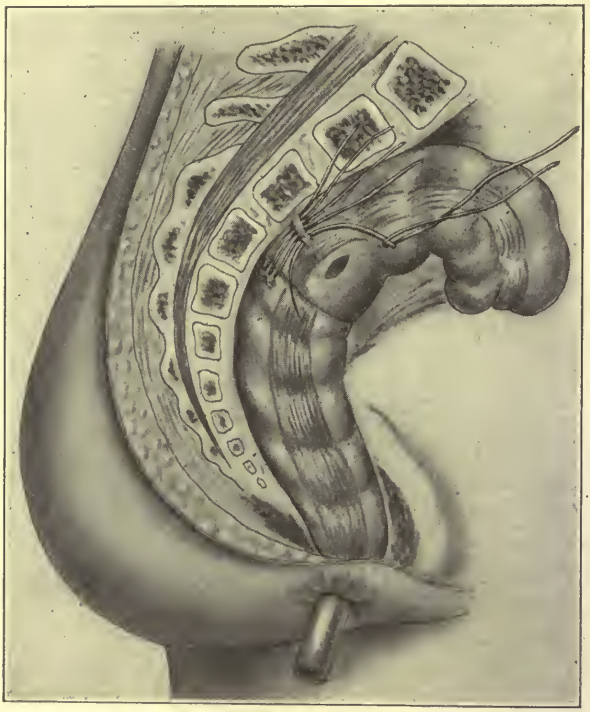


FIG. 899.— SIGMOIDORECTAL INVAGINATION (Morrison's Method).

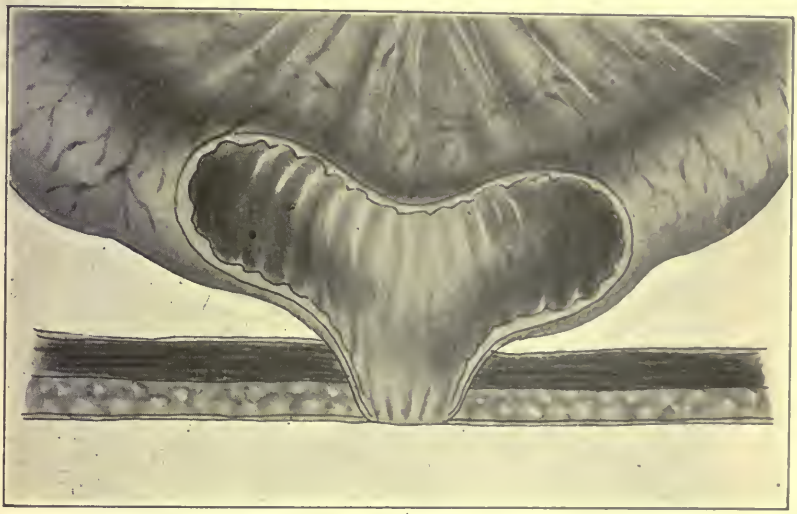
The intussusception is held in place by suture.

"5. A ligature is then passed around, immediately below the lower cut end, and tightly tied. This makes the junction water tight.

"6. The tube is again pulled upon, whilst the lower portion of bowel immediately below the button is steadied, till, by traction from above, a short intussusception is produced. This is maintained by a few Lembert sutures.

"In from four to eight days the ligatured sloughing bowel separates,

and the tube is then released. I have performed this operation fourteen times; twice for complicated pyosalpinx, in the remaining twelve cases for malignant disease."



A

B

FIG. 900.—*A*, ARTIFICIAL ANUS WITHOUT A SPUR; *B*, WITH A SPUR.

Figure 899 gives an adequate idea of the condition of affairs when the operation is completed.

Removal of growths causing obstruction of the gut is best accomplished by an operative method which obviates the mortality attendant upon immediate suture of the intestine. According to Mikulicz,[38] the high mortality is caused mainly by the secondary changes which take place in the gut under the influence of carcinomatous stenosis. The gut is dilated, its nutrition is impaired, and its muscular wall is insufficient. Consequently, after the operation the atonic condition of the intestine allows arrest of the contents at the site of suture, which

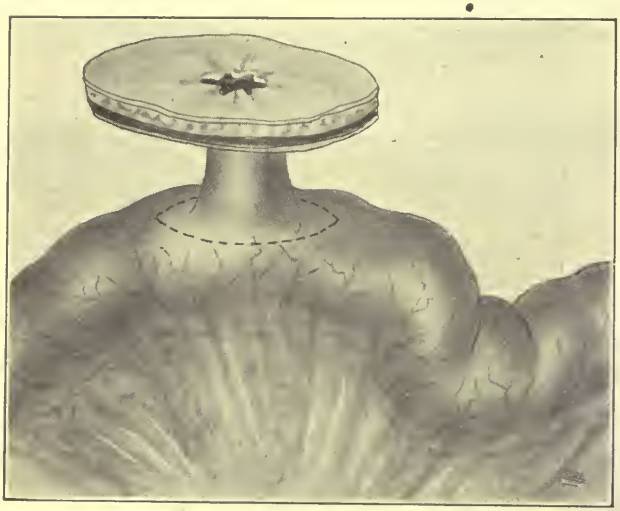


FIG. 901.— FECAL FISTULA.

The dotted line shows the portion of gut wall excised.

yields, and peritonitis results. Mikulicz[38] says "For a number of years I have invariably performed the operation in two sittings and have found that the results were infinitely better than formerly. The primary incision, the enucleation of the tumor, the removal of the lymphatic glands — in short, the entire operation — is performed exactly as when one operation only is done. If, now, the tumor has been freed and completely enucleated, it is drawn out of the wound, the loop of gut is stitched to the parietal peritoneum with sutures including only the serous coat, and the abdominal wound is closed, leaving only room enough for the loop of gut. Now, only after the

abdominal eavity is completely elosed, the tumor is excised, and an artificial anus is established, whieh is closed in from two to four weeks, aceording to the usual methods. The disadvantage of this process is that the patient is afflieted for a few weeks with an artificial anus, but I believe that this is fully balanced by the advantage of greater safety."

Openings between the intestine and the surface of the skin (*fecal fistula and artificial anus*) are of three kinds:

1. A small opening in the skin leading to an opening in the gut.

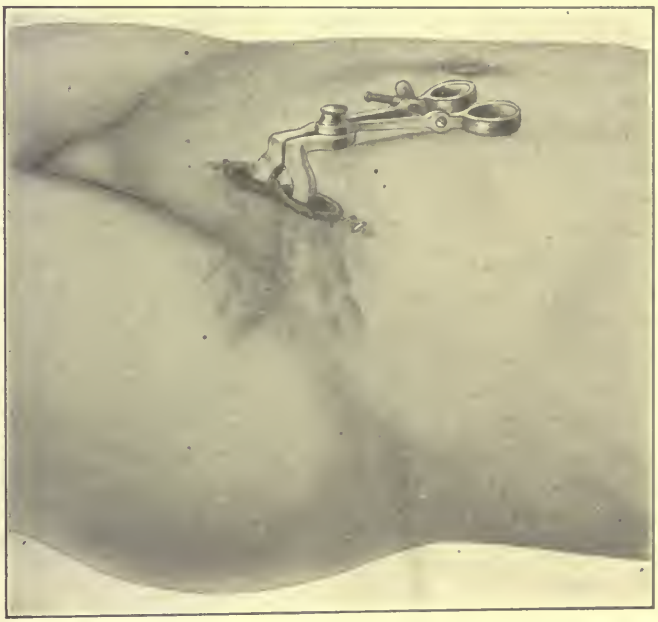


Fig. 902.— Enterotome Crushing through Wall of Large Gut.

2. A large opening in the gut, the mucosa of which is adherent to the skin. The mucosa of the opposite side of the bowel is visible through the opening (Fig. 900A).

3. The intestine, as it comes in contact with the surfaee, has two limbs on either side of a *spur* (Fig. 900B).

In the *first type* the following operation is most satisfactory: An elliptieal incision is made about ½ inch from the margin of the skin opening and earried through the subeutaneous tissues and museular layer of the abdominal wall to the peritoneum. The peritoneum is

then opened, the loop of gut delivered through the wound with the ellipse of superficial tissues, and the fistulous tract attacked. The latter is removed, together with an oval sector of the intestinal wall (Fig. 901). The opening left in the bowel is closed, at right angles to the long axis of the gut, with a double layer of sutures.

In the *second variety* of fistula it is best to separate the gut from the abdominal wall by careful dissection, and close the opening by transverse sutures as stated above. If there has been much loss of substance, the corresponding area of gut is resected and the continuity of the bowel re-established by end to end anastomosis.

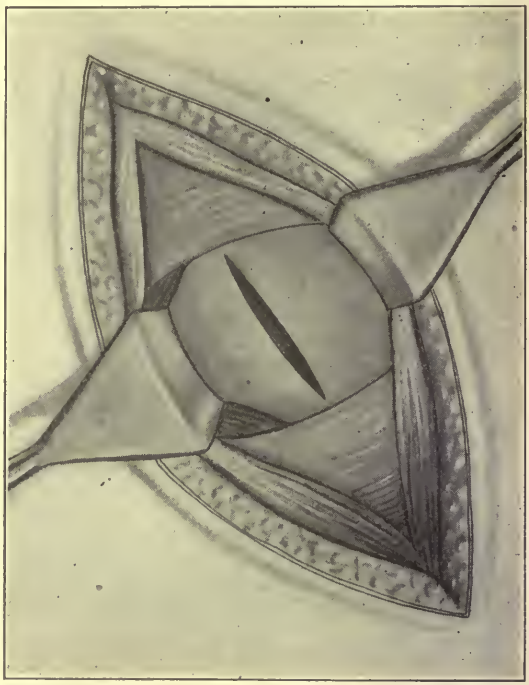


FIG. 903.— HARRINGTON'S INTERMUSCULAR APPROACH FOR APPENDECTOMY.

The rectus muscle is drawn toward the median line.

In the *third form* of fistula, that is, when a spur is present, resection of the involved portion is often necessary. If the conditions in this connection present difficulties, the use of the *enterotome* is permissible. The instrument consists of a pair of forceps with long, roughly, serrated blades, which are pressed together and held by a clip or a screw handle. One blade of the instrument is introduced into the upper opening and the other into the lower, so that when the blades are closed, the spur is grasped between them. The pressure of the blades is tightened daily until the spur is destroyed. Spontaneous closure of the fistula usually follows the maneuver.

Appendectomy.— Removal of the appendix is accomplished by two

methods (1) the intermuscular; (2) by an incision which displaces the rectus muscle to the inner side. Most surgeons prefer the latter.

The intermuscular incision (McBurney[39]), modified by Harrington,[40] is described by the latter as follows:

"Make an incision in the skin 4 to 5 inches long in a line with the fibers of the external oblique muscles, at such a distance from the anterior spine of the ilium as is desirable. Separate the muscle and its tendon for a like distance, then insert retractors 1½ inches broad at either end. The middle of the incision should correspond to the usual position of the base of the appendix. At this point the internal oblique and transversalis muscles run nearly parallel and horizontal. These muscles should be separated without cutting down to the transversalis fascia, and from the ilium to the linea semilunaris. If this does not give sufficient room, the sheath of the rectus may be separated in front and behind the muscle in a line continuous with the separation of the internal oblique and transversalis muscles.

"The sheath of the rectus muscle is formed by the aponeuroses of the internal oblique and transversalis muscles. The direction of the fibers of the sheath is horizontal, being at right angles to the muscular fibers of the rectus. At the semilunaris line it will be necessary to use the knife or scissors, since at this point the union of the various aponeuroses is very compact. The rectus muscle can then be drawn toward the linea alba, and considerable room obtained.

"Care should be taken not to cut the deep epigastric artery, which at this point lies inside the rectus sheath in the posterior part of the muscle. If necessary, it may be tied. Two retractors, each about 2 inches broad, should be inserted, one at either end of the deep incision; the retractors which have been used for the external oblique can now be dispensed with."

The cecum is identified, delivered into the abdominal wound, and the longitudinal muscular band followed until the appendix is reached. If stripping of adhesions is necessary, the operative field is isolated by means of gauze pads. The appendix and the adjacent part of the cecum are delivered from the abdominal cavity. An assistant wraps the appendix around with gauze. An artery clip is then used to transfix the meso-appendix, about 1 inch from its apex, to seize a catgut ligature, and is then withdrawn. The ligature is tied and the meso-appendix divided. This proeedure is repeated until the base of the appendix is reached. The appendix is then held vertically by means of a clip, whilst the surgeon crushes it in a clamp at its junction

with the cecum. At this point a fine ligature is applied (Fig. 904). The appendix is divided just distal to the ligature. It is important to remove all of the appendix. The stump may be buried in the wall of the cecum by a row of Lembert sutures, or a purse string suture may be employed for the purpose.

As already stated, appendectomy is most frequently performed through the Battle[41] incision, which the originator of the method describes as follows:

"An incision is made through the skin and subcutaneous tissues down to the sheath of the rectus; this section is made obliquely downwards midway between the anterior superior iliac spine and the um-

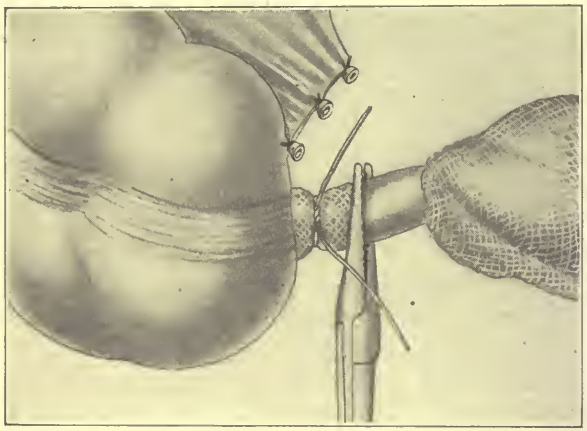


FIG. 904.— APPENDECTOMY.
The base of the appendix is crushed and ligated.

bilicus, is about 4 inches in length, varying with the deposit of fat in the abdominal wall of the individual, and is placed so that it is equally extended above and below this line. The outer margin of the rectus sheath can now be easily defined, and during inspiration the direction of the action of the external oblique aponeurosis seen. The inner margin of the wound is retracted about ½ inch, and the anterior sheath of the rectus opened close to the retracted inner margin for the full extent of the incision. The rectus muscle is now drawn inwards, being separated from the outer part of the sheath with a few touches of the knife. Occasionally one of the lineae transversae requires to be separated from the sheath. Running across the posterior layer of the sheath can usually be seen the dorsal nerves, with accompanying ves-

sels. The deep epigastric artery and vein run upwards under the retracted muscle to the inner side, and it is well to define these vessels, otherwise the vein may be punctured in the later suturing of the sheath. Another anatomical point of importance is the level of the transversalis fascia as regards the wound, for it strengthens the posterior layer of the sheath and makes the hold of the sutures much firmer, if the opening into the peritoneal cavity can be confined to the part covered by it. In most instances the incision can be made through the posterior sheath between the nerves and through the fascia. We

FIG. 905.— APPENDICOSTOMY.

have not, however, seen any ill effects from division of these nerves, although it has been suggested that partial atrophy or paralysis of the rectus muscle might ensue. If the case is uncomplicated by adhesions and the appendix is lying in a normal position, the operation can be completed through an inch long incision of the posterior sheath; but if there are complications and it is considered necessary to do so, the wound, both superficial and deep, can be extended to any required distance. After the appendix has been removed by the method which is considered best, and the peritoneum cleansed, the posterior layer of the sheath and peritoneum can be sutured in the usual manner. The

uninjured rectus is now permitted to fall back into its usual bed; the anterior layer of the sheath is closed by 3 or 6 interrupted (.00) silk sutures, and the skin and superficial wound by a continuous horsehair stitch. Union takes place throughout by first intention, and the horsehair stitch is removed on the eighth day."

In cases of *abscesses developing in connection with the appendix* the incision is made over its most projecting or superficial point. The usual incision for appendectomy is by no means always the proper one for the opening of an abscess. After the abscess is defined the

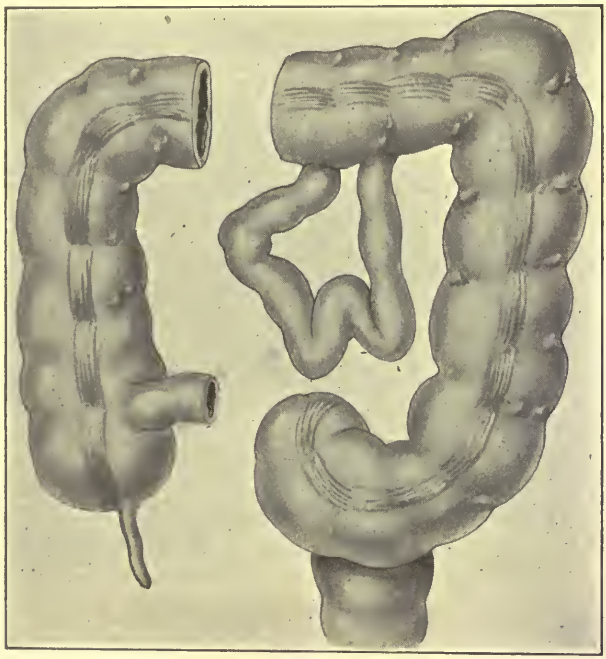


FIG. 906.— BILATERAL EXCLUSION.
Both ends of excluded gut are closed and the sector is drained through a fistula.

abdominal wall is carefully opened, layer by layer, until, when the edematous peritoneum is opened, pus is discharged. The finger is gently introduced and the limits of the abscess defined. It is expecially important that the lower limit of the cavity be recognized and, if necessary, a second incision be made in this location for drainage. IF THE APPENDIX LIES WITHIN THE ABSCESS CAVITY IT IS REMOVED; IF NOT, IT IS NOT SEARCHED FOR. The cavity is now gently swabbed out and

tube drainage established. This should be maintained for from 6 to 8 days, the patient being postured so that gravity is aided.

Appendicostomy for drainage of the large intestine was suggested by Keetley,[42] and later popularized by Wier.[43] The measure is employed to permit of the introduction of solutions into the colon, in the treatment of mucous colitis, dysentery, etc.

The appendix is exposed through an intermuscular incision (p. 1957),

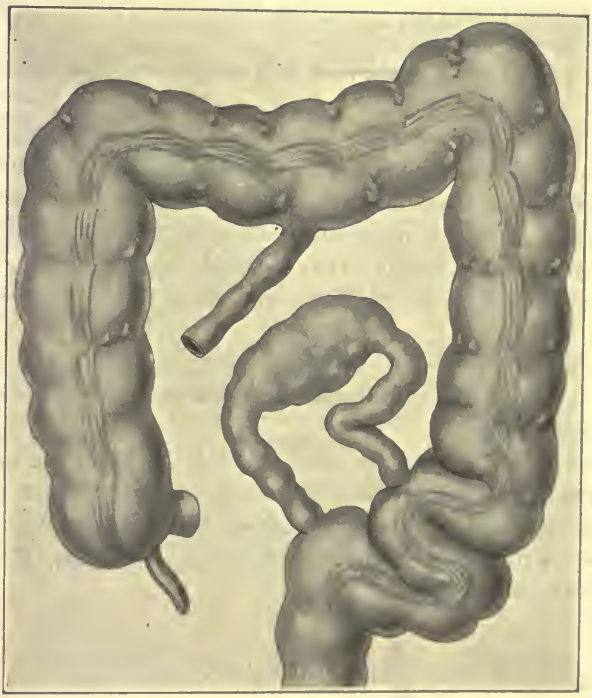


FIG. 907.— INTESTINAL EXCLUSION.
Both ends of excluded segment are joined to the sigmoid.

delivered through it, and fastened to the fibers of the external oblique muscle, as shown in Fig. 905.

Intestinal Perforation.— In intestinal perforations the damaged loop is delivered through the celiotomy wound and isolated by gauze packing. If the opening is small, a purse string suture may be used to close it and this buried by one or two Lembert stitches. Large perforations are treated by the temporary establishment of a fecal fistula, which is later closed in the manner described above (p. 1955).

Intestinal Exclusion.— Intestinal exclusion is a term applied to the art of rendering inactive any part of the intestinal canal by surgical means.

In exclusion by *simple entero-anastomosis,* as suggested by Maisonneuve,[44] the fecal current is not entirely diverted, and the object is not attained.

In *entero-anastomosis and closure of the upper opening by suture,* the fecal current passes in the reverse direction into the isolated loop. Thus the object is not accomplished.

Complete exclusion is performed by the *bilateral* and the *unilateral* methods.

In *bilateral exclusion* the gut is divided at two places and the intestinal channel restored, leaving a segment of the bowel detached. Of the excluded segment one end may be left open and the other closed; or both ends may be left open and stitched to the skin; or both ends may be closed when a fistula exists between them; or both ends may be united to a segment of intestine, into which the loop between them will drain.

In *unilateral exclusion* the gut is divided completely across and the proximal end implanted into the side of the distal end. The latter may be closed or left open.

Exclusion of the intestine is practiced for the following conditions: Tuberculous or simple ulcers of the gut, which have led to the formation of external fistula; intestinovaginal fistula; artificial anus and fecal fistula; colitis and chronic intractable constipation (Hartmann,[45] Narath,[46] Moynihan,[1] Lance[47]).

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CHAPTER IV

INJURIES OF THE STOMACH AND GUT, FOREIGN BODIES, GASTRIC AND INTESTINAL FISTULAE

Injuries of the Stomach and Gut.— Subcutaneous injuries of the stomach and gut are the result of blunt force, such as a kick, passage of a wheel, fall on a hard substance, etc. As a rule, the organ immediately below the site of the injury is affected. However, this is not always the case; occasionally the force and its effect are transmitted to a distal organ.

The *mechanism* by which the stomach and gut are injured is divided into *crushing, avulsion by tearing,* and *bursting.*

Crushing is the result of circumscribed force or of the organ being compressed against the vertebral column.

Avulsion and bursting occur in connection with hypertension of the organ beyond the limit of its elasticity. The solution of continuity does not necessarily correspond to the location where the provocative force is applied. *Avulsion* occurs at the site of attachment of the organ. The stomach is rarely avulsed. On the other hand, the gut is not uncommonly torn away from the duodenojejunal or the ileocecal fossae, and at the flexures of the colon. *Bursting* happens when a sector of gut is filled with gas or fluid and both ends of a loop of gut are compressed by the provocative force. It is probable that comparatively moderate force directed against a distended sector of gut may cause it to burst.

Injuries of the wall of the stomach and gut vary in degree. They are classified as *contusion, non-perforating* and *perforating.*

A *contusion* may be so slight as to escape notice. In other instances the injury is attended with excessive hemorrhagic infiltration followed by necrosis, while in others a more or less extensive hematoma is formed. A restricted area of necrosis of the mucosa is followed by ulceration (traumatic ulcer).

Non-perforating lesions consist in tearing or contusion of a part of the visceral wall. These may involve one or two walls of the organ.

Perforating lesions are the result of avulsion or bursting injuries.

Contusions and non-perforating injuries of the stomach are attended with the clinical picture of shock. The presence of blood in the vomitus and stool, following injury in the epigastric region, without the rapid development of evidence of peritoneal irritation justifies the diagnosis. The treatment is purely symptomatic.

Perforating lesions of the stomach (usually rupture) occur most frequently at the pylorus or lesser curvature. As a rule, other organs (liver, spleen, pancreas) are also injured. No doubt the presence of a pathological condition, such as an ulcer, favors traumatic rupture of the stomach.

The *clinical picture of rupture of the stomach or gut* is, in most instances, characteristic. The symptoms are divisible into those of *shock*, those appearing *at the site of the perforation*, and those indicating involvement of the *peritoneum as a whole*. Unless the patient is unconscious, the initial *pain* is very severe and is at first localized at the site of the perforation. Shock is likely to be profound and often merges into coma. *Vomiting* is frequent, but, strange to say, occurs oftener in intestinal than in gastric perforation. *Rigidity of the abdomen develops early*, and when it persists for several hours may be regarded as indicating perforation of the stomach or gut. The subsequent course of the affection corresponds to the clinical picture of peritonitis (p. 1890).

The persistence of shock and the absence of the signs and symptoms of peritonitis indicate intra-abdominal bleeding. The establishment of the presence of free gas in the abdominal cavity is pathognomonic of rupture. However, this is not always easy to determine, especially when liver flatness is abolished as the outcome of meteoric distention of the gut. In some instances loops of distended gut are forced in front of the liver. An effort should be made to ascertain the location of the perforation by a study of the site and character of the causative force.

Late perforation is characterized by an interval between the picture of shock immediately sequential to the injury and the development of the evidences of peritoneal irritation. The interval may be due to temporary occlusion of the perforation by the prolapsed mucosa; or the perforation may become agglutinated to a contiguous organ and later permit discharge of the visceral content; or an area of the visceral wall may be severely contused and subsequent perforation may occur as the outcome of sloughing. There is no doubt that occasionally perforation of the gastro-intestinal canal is not attended with

leakage of the contents and that spontaneous closure of the opening may occur. However, this desirable outcome is exceedingly rare. In addition to this, a perforation may become circumscribed by protective adherence of the omentum and other parts contiguous to the lesion and an abscess may develop; or an extraperitoneal perforation, especially of the colon, may terminate in abscess formation. But these contingencies too are very rare.

In the vast majority of cases the *prognosis* of rupture of the stomach and gut is unfavorable, unless operative measures of relief are promptly employed. This is true even in those cases localized by adhesions, in which ultimately invasion of the general peritoneal sac is almost certain to occur.

The *treatment of acute perforation of the stomach and gut* is the same as that of peritonitis, plus repair of the lesion, both of which have already been fully discussed. The best time to operate is between the shock and the development of peritonitis (Kausch[1]). However, if shock persists, the presence of progressive internal bleeding may be assumed and the operative measures must not be delayed.

Penetrating wounds of the abdomen are usually attended with *perforation* of the contained organs and are the result of stab and gunshot wounds, including injuries with more or less pointed objects.

If the wound in the abdominal wall is large, prolapse of the gut or omentum is likely to occur. The prolapsed organ is not always the injured one.

Perforating wounds produced by sharp instruments rarely involve more than one sector of the gut, while gunshot wounds are likely to be multiple. The number of perforations depends upon the angle at which the projectile traverses the abdomen.

The *clinical picture* of penetrating wounds of the gut and stomach does not differ from that described in connection with subcutaneous injuries, except with regard to the occasional prolapse of the gut or omentum in the former.

The *diagnosis,* on the whole, is made easier than in subcutaneous perforations by a study of the wound in the abdominal wall, as to its location and character. As in subcutaneous injuries, the persistence of shock indicates bleeding from an injured blood vessel. From the standpoint of experience, one is justified in stating that IN ALL CASES IN WHICH THE CIRCUMSTANCES OF THE INJURY POINT TO THE PROBABILITY OF INJURY OF THE STOMACH AND GUT THE ABDOMEN SHOULD BE OPENED.

As in subcutaneous injuries, the *course* of perforating wounds of the stomach and gut, unless repaired by operative means, is exceedingly unfavorable. The almost always fatal outcome is the result of peritonitis and internal bleeding. The latter does not come from the walls of the injured viscus, but from the vessels of the mesentery. In this class of cases temporary inflammatory coalescence of contiguous parts may cause an illusionary lull in the symptoms, to be followed by sudden extension of the process and fatal general peritonitis (Treves,[2] Makins,[3] Abadie[4]).

The so-called *conservative treatment* of perforating wounds of the gastro-intestinal canal consists in keeping the patient quiet and in the administration of opiates. There is no objection to an occasional prayer.

The *operative treatment* consists in celiotomy and repair of the injured viscus (chap. iii). In stab wounds the abdomen may be opened at the site of the injury; in gunshot wounds median celiotomy is preferable.

Injuries of the stomach from within are most frequently caused by caustic agents. The resultant damage usually occurs at the oral end of the lesser curvature and at the pylorus.

The immediate *symptoms* are pain, tenderness, and vomiting. The vomitus usually contains blood and, at times, shreds of necrotic mucosa. Shock is often severe and may be fatal. The *diagnosis* is based on the history of the case and on the appearance of the vomitus.

Moderate cauterization is followed by complete restitution to the normal; more severe lesions are followed by cicatrization and deformation of the stomach. The degree of deformity varies, i. e., pyloric obstruction, shrinkage of the entire stomach, and hourglass stomach. Chronic ulceration is a common sequel.

Primarily the *treatment* is directed toward chemical neutralization of the caustic agent. Gastric lavage is contra-indicated in severe cases because of the danger of perforation of the esophagus or stomach. It may be employed for the purpose of removing carbolic acid or corrosive sublimate. The subsequent treatment aims at administering non-irritating food.

Surgical measures are indicated in the following:

1. Perforation and peritonitis demand celiotomy, repair of the stomach, and cleansing and drainage of the peritoneum.

2. Extensive destruction of the gastric mucosa justifies the employment of *jejunostomy;* great cicatricial deformation brings up the

question of *gastro-enterostomy*. If the esophagus is strictured, *gastrostomy* may be necessary.

3. Pyloric stenosis calls for gastro-enterostomy or pyloroplasty; hourglass stomach for *gastrogastrostomy*.

4. Stenosis of the cardia may be treated by bougie dilatation (p. 1788).

Foreign Bodies.— Foreign bodies in the stomach are usually introduced in the natural way. Their introduction may be accidental or, in the case of professional trick performers, they may be intentionally swallowed. In addition to this, gallstones occasionally find their way into the stomach, especially when the duodenum is obstructed as the result of cicatricial contraction; or the gallbladder may become adherent to the stomach, perforate into it, and the stones gain access through the fistulous opening thus established. The habitual swallowing of hair is followed by the formation of a "hair ball" in the stomach. This occurs most often in young girls who have the habit of gnawing their hair braids, etc. Occasionally a hair ball of this sort fills the entire lumen of the stomach.

The consequences of the introduction of foreign bodies into the stomach vary with the size and conformation of the object. Small bodies, unless they possess sharp edges, do not cause serious disturbances. Needles are especially likely to perforate the stomach wall and provoke the picture of gastric perforation. On the other hand, foreign bodies may remain in the stomach for a long time without causing disturbance.

The *diagnosis*, in the absence of an indicative history, is likely to be extremely difficult and the clinical picture very apt to be that of chronic gastric ulcer, in which event the Röntgenographic findings are of great value.

The ultimate fate of the foreign body depends upon its character; a number of smaller objects are likely to pass out of the pylorus and be discharged per rectum. Occasionally a body is incarcerated in the pylorus or is forced into the small gut and causes obstruction in this situation.

When the presence of a foreign body in the stomach is established, the *treatment* depends upon the character and dimensions of the unwelcome visitor. If the body is small, a diet of potatoes or grain gruels may be given and the result awaited (catharsis is contra-indicated). Immediate operative measures are indicated only when symptoms of perforation develop. Larger bodies are best removed by *gastrotomy*.

Foreign bodies in the gut are propelled onward from the stomach or enter from the peritoneal cavity (left in by the surgeon). In the latter event, the body ulcerates into the gut within an area of local peritonitis. Substances introduced into the rectum rarely ascend higher than the lower end of the sigmoid flexure. In addition to this, certain bodies are found within the gut from the constituents of the normal contents as follows:

1. *Enteroliths* are genuine concretions and consist of salts (phosphorus, lime, and magnesia) which encrust a small nucleus.
2. *Coproliths,* which consist of inspissated feces and may assume great size.
3. *Fecal tumors,* which consist of kneadable masses of feces; these develop in the colon and rectum.

Occasionally accumulated seeds, plant fibers, worms, etc., form a large mass in the gut.

Foreign bodies that are propelled through the pylorus are rarely large enough to obstruct the bowel. They are most likely to be arrested in the flexures of the duodenum (Jaennel[5]), rarely in the large intestine. On the other hand, even small bodies may become incarcerated within a zone of intestinal stenosis.

The passage of a foreign body through the gut often occurs without *symptoms.* The arrest of a body is attended with the clinical picture of intestinal obstruction. If ulceration and perforation occur, the picture is that of circumscribed or diffuse peritonitis.

The *diagnosis* is not difficult if an indicative history is available. If the latter is not obtainable, the diagnosis will be concerned with recognition of intestinal obstruction or of the complicating peritonitis, which will, in all probability, not be elucidated until the operation. Fecal tumors, on the other hand, are frequently recognized by their doughy consistence, elicited upon palpation of the abdomen.

The *treatment* of foreign bodies consists in enterotomy and subsequent suture of the opening.

Gastric Fistulae.— Gastric fistulae, other than the operative, are very rare. They occur in connection with penetrating and subcutaneous wounds of the stomach. A fistula may develop immediately after the injury or may be preceded by a brief period of circumscribed peritonitis which perforates the anterior abdominal wall. Occasionally a gastric ulcer becomes adherent to the abdominal wall and perforates (Lieblein and Hilgenreiner[6]).

The *diagnosis* is made by recognition of the character of the fistulous

discharge. If the fistula is very small, the acidity of the gastric juice may be neutralized by the wound secretions.

Small fistulae often heal spontaneously. This is illustrated in connection with gastrostomy in which the opening closes soon after the rubber tube is permanently removed. Larger fistulae are readily repaired by excision of the adventitious tract and by closure of the opening of the stomach by means of a double row of Lembert sutures.

Intestinal Fistulae.— Intestinal fistulae (fecal fistulae) occur in the following ways:

1. From *penetrating wounds* of the abdomen with prolapse of the gut, which becomes adherent to the peritoneum and perforates; or when extravasated feces cause localized peritonitis, which subsequently ruptures through the abdominal wall. Subcutaneous injury of the gut may result in the establishment of a fecal fistula in a similar manner.

2. When a fecal fistula is *intentionally* established for the purpose of draining the intestinal canal, or is *unintentionally* caused by accidental injury to the gut during an operation.

3. In connection with *strangulated hernia* in which the gut becomes gangrenous and perforates.

4. As the result of *ulcerative processes* of the gut (tuberculosis, carcinoma, actinomycosis, peptic ulcer) which perforate the abdominal wall.

5. When pathological conditions of the peritoneum, such as tuberculosis, simultaneously invade the gut and the abdominal wall and cause perforation of both, so that a communication is established between the interior of the canal and the surface of the body.

Fecal fistulae develop most frequently in locations where the etiological diseases most often originate, i. e., in the ileocecal and the inguinal and umbilical regions.

Intestinal fistulae are *direct* and *indirect*. The fistulous tract may be lined with intestinal mucosa or with dermal epithelium. The former is the rule.

An intestinal fistula differs from an artificial anus in that in the former only a part of the intestinal contents is discharged through the opening, while in the latter all of it finds egress through the preternatural avenue. Between these, various degrees of fistulae may be found.

The *conformation and cause of a fecal fistula* is as follows: if the intestinal loop communicating with the fistula is not kinked, its contents moves in the normal peristaltic direction and the loop (aided

by the mesentery) tends to withdraw itself from the adventitious opening. In this way the gut may succeed ultimately in freeing itself from where it is adherent to the abdominal wall; the portion of the gut attached to the opening is elongated and the granulating fistulous tract heals. If the fistulous canal is lined with mucosa (lip fistula) it cannot heal. The gut is drawn away from the opening, but a funnel shaped portion remains attached to the fistula. In this way the angle between the oral and aboral segments of the gut is made acute, so that the two lumina ultimately lie parallel to each other; the two apposed walls of the gut lie in contact and a *spur* is formed. The spur corresponds to the mesenteric attachment of the gut. As time goes on, peristalsis endeavors to force the oral sector out through the fistula, while the aboral portion is drawn away from the opening. Finally, the intra-abdominal pressure causes the mucosa of the gut to prolapse through the opening and, if the latter is large, the entire oral portion of the loop is extruded. In this event, the prolapsed sector of gut may become strangulated.

The higher an intestinal fistula is located the more it interferes with the nutrition of the host, and the more severe is the effect upon the surrounding skin.

The *diagnosis* of intestinal fistula is usually self evident. Occasionally (in small fistulae) a microscopical examination of the discharge is necessary. It is important to determine the location of the fistula. While microscopical examination, time tests, and the like are valuable in this connection, the weight of the patient may be regarded as a safe clinical guide as to whether delay in the employment of operative measures of relief is justified.

The *operative treatment* of intestinal fistula has already been taken up (p. 1970).

Internal intestinal fistulae consist in abnormal communication between the gut and other hollow organs. They are most likely to occur between organs which normally communicate with the intestines (gastro-intestinal, ileocecal, etc.); the biliary passages are often connected with the gut by fistulous tracts. In addition to this, the intestinal tract may establish fistulous communication with the kidney, the ureter, the bladder, and the female organs of generation.

All internal fistulae are the result of the same factors discussed in connection with external fistulae, the provocative process (tuberculosis, carcinoma, etc.) originating either in the gut or in the communicating organ. For instance, while an ulcerative lesion of the gut may

perforate into the bladder, a similar process in the latter may perforate into the gut.

The *treatment* of intestinal fistulae consists in exposure of the tract, repair of the openings, and if necessary (and feasible), extirpation of the offending organ or part.

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CHAPTER V

METHODS OF EXAMINING THE STOMACH AND GUT

Inspection takes cognizance of the general conformation of the abdomen, general meteorism, dilated veins, the type of respiration, and local swelling. Very often the eye detects the contour of the stomach, the outline of intestinal loops, and the presence of a tumor. The motility of the latter with regard to respiration is also noted. Finer distinctions with regard to the location of a tumor are best made by viewing the abdomen from a distance.

Palpation is of great aid to the surgeon. For the purpose the patient is postured in the supine position with the head slightly flexed and the thighs brought to the horizontal position. One hand "feels" while the other, superimposed upon it, makes pressure. Light pressure is more discerning than heavy. The measure outlines tumors and standardizes resistance. In palpating a tumor the hand notes its size, form, character of surface, consistence, its active or passive motility, whether it is tender or not, and its relationship to anatomical landmarks. In the lower sector of the abdomen (pelvis), digital examination per vaginam and rectum are very important.

Percussion assists in the detection of exudates, tympanic areas, extent of liver dullness, etc.

Auscultation is used to determine the presence of intestinal gurgling, the movement of gases, gastric succussion and peritoneal friction.

Inflation of the stomach and large gut is best accomplished with a rubber tube, using air for the purpose. In this way the outlines of the stomach and colon as far as the cecum are easily recognized. *Water* may be used in the same manner.

Gastroscopy is of value in determining the character of gastric lesions located near the cardia. It has a limited field of usefulness.

Röntgenography has become an exceedingly useful aid to diagnosis Its significance is taken up with the particular conditions.

Rectoscopy and *sigmoidoscopy* have advanced almost to an exact science. The measures make clearly visible the interior of the rectum

and sigmoid. In some instances it is possible to visualize the descending colon (Kemp,[1] Tuttle[2]).

Chemical and microscopical examination of the stomach contents and similar examinations of the excreta are of corroborative diagnostic value.

A description of the laboratory methods does not belong here. However, it is perhaps proper to call attention to the fact that an obscure condition is often illuminated by employment of the methods available in this connection.

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CHAPTER VI

PYLORIC STENOSIS, HOURGLASS STOMACH, CONGENITAL ANOMALIES OF THE STOMACH

Pyloric Stenosis.— Pyloric stenosis presents a symptom complex which is independent of its underlying cause. It is caused by *cicatricial contraction, ulceration,* and *tumors* (usually carcinoma), and occasionally by kinking, adhesions, pressure from without, gastroptosis, pathological contraction (pylorospasm), congenital anomalies, and, finally, by a foreign body. Two forms of pyloric spasm must be differentiated, namely, the purely neurotic and that dependent upon an organic lesion. The latter is due to ulcer, fissure, and varicosities of the veins of the pylorus. The *course* of the affection is essentially chronic.

The *symptoms* of pyloric stenosis are at first those of so-called *dyspepsia* — loss of appetite, nausea, coated tongue, eructations, etc. Dyspepsia is caused by delay in emptying the stomach, by retention of food, and by the chronic catarrhal condition of the stomach incidental to a lesion in this situation (ulcer, etc.). *Pain* is not a marked symptom. On the other hand, a feeling of pressure and discomfort is usual. *Vomiting* always occurs in advanced stenosis of the pylorus, and is usually present in the milder forms. It is ascribable to overdistention and to irritation caused by the presence of an ulcer. The expulsion of particles of diet taken the previous day is characteristic. Pyloric obstruction without *motor disturbances* is inconceivable; *per contra,* the absence of delay in emptying the stomach excludes pyloric stenosis. If the stomach is not emptied at the end of ten or twelve hours, the *insufficiency* is that of the *second degree;* moderate delay is designated as *insufficiency* of the *first degree.*

The motor disturbances depend upon the degree of stenosis and the force of the gastric musculature. The latter often undergoes secondary atony, especially in carcinoma. Overdistention of the stomach ultimately leads to *megalogastria.* However, a small infiltrated stomach may exhibit a distinct insufficiency, and a large one may show

none (diabetes). A dilated stomach (*ectasia*) usually sinks downward (*gastroptosis*). This is likely to give rise to splashing sounds.

Acceleration of peristalsis always occurs in pyloric stenosis. This is readily demonstrated by percussion of the epigastrium and is both palpable and visible. Occasionally the maneuver produces a sustained rigid contraction of the stomach, which gradually relaxes (gastric rigidity) beneath the palpating hand.

The increased activity of the stomach leads primarily to hypertrophy; later, the musculature is relaxed and atony ensues. Demonstrable increase in peristalsis points strongly to pyloric obstruction.

The stenosis itself is rarely palpable. Analysis of the gastric secretion shows wide variations, which are more indicative of the underlying causative factor than of the existence of obstruction. Bacterial flora is increased.

The *systemic effect* of pyloric obstruction is that of *malnutrition.* Only that portion of the food which passes the pylorus is absorbed; the retained portion undergoes fermentation and decomposition and little of it is taken up by the blood. The latter is attended with a peculiar *tetany* ascribed to auto-intoxication. Anhydremia, the result of lessened absorption of fluids, is a constant symptom of pyloric obstruction, and is increased by the greater secretory activity of the stomach compared with its capacity to absorb fluids. This is responsible for *lessening of the quantity of urine and stool.*

The *diagnosis* is based on the symptoms stated, of which some are determining. Of these, retention of food (over night) is the most certain. Of the various diagnostic measures, the Röntgenographic findings are the most valuable. From the practical standpoint, the prognosis and treatment are influenced more by the severity of the functional disturbances, than by the determination of the degree of stenosis by refinements in diagnostic measures. It is also exceedingly important to differentiate pyloric stenosis, due to a neurosis, from the mechanical, especially with regard to treatment. Gastro-enterostomy has been performed more than once for the relief of symptoms attendant upon beginning tabes dorsalis. It must be remembered that pylorospasm is not accompanied by clearly defined motor disturbances.

With regard to *prognosis* and the decision regarding *treatment,* the surgeon must differentiate sharply between pyloric stenosis with slight motor disturbances and that with severe motor disturbances. The former causes little damage to the general condition of the patient, while the latter presents a grave menace in this connection, which is

not obviated by the use of gastric lavage, diet, etc. In severe cases the question of surgical efforts at relief is easily answered in the affirmative; in the others the answer is not so easily given. However, when there is the least suspicion of carcinoma, there is, of course, no doubt that operative measures are distinctly indicated.

The *surgical treatment* is directed toward removal of the lesion (chap. iii of this part). If this is not feasible, gastro-enterostomy must be resorted to. In cases of pure cicatricial stenosis, *pyloroplasty* is the operation of choice, on the ground that it preserves the physiological relationship of the parts. It is by no means certain that the unrestrained discharge of gastric contents directly into the jejunum is without its disadvantages — a viewpoint the writer is inclined to take. There is no doubt that gastro-enterostomy has an unfavorable effect upon pylorospasm due to nervous influences.

Hourglass Stomach.— *Hourglass stomach* consists in stenosis of the body of the stomach. It is usually situated near its middle (Moynihan[1]). The condition is, in a general way, produced by the same factors which enter into the causation of pyloric stenosis, and produces a similar clinical picture. The *diagnosis* is so readily established by the *Röntgenographic findings* that farther discussion in this connection is superfluous.

The *indications for operation* are the same as in pyloric stenosis. The methods of procedure are sectional resection of the stomach, gastro-enterostomy (cardiac sac), and gastrogastrostomy. The last is the operation of choice.

Congenital Anomalies.— Of the congenital anomalies of the stomach, pyloric stenosis is the only one which possesses surgical importance. In a restricted number of cases, the obstruction is due to contraction of the pylorus. However, in most instances the lesion consists in a genuine hypertrophy of the musculature of the pars pylorica.

The *symptoms* develop within the first year of life and closely resemble those of the adult, but are more rapidly followed by atony of the stomach and death. Hyperacidity is absent; the gastric juice of the nursing infant does not contain hydrochloric acid. As mechanical stenosis cannot be differentiated from spasmodic stenosis, an effort may be made to correct the condition by diet, etc. If this fails, pyloroplasty is indicated. The operation is usually well borne (Trantenroth,[2] Schotten[3]). In this connection, the work of Downes[4] is particularly illuminating.

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CHAPTER VII

ULCER OF THE STOMACH AND DUODENUM

Ulcer of the Stomach.— Ulcer of the stomach may be characterized as an ulcerating defect (a necrosis) of the mucosa or of the mucosa and the deeper structures of the stomach wall. It shows little tendency to heal, but may remain stationary for years, or may extend. Kausch[1] differentiates between *simple* (ulcus rotundum, chronicum, pepticum) and *traumatic ulcer*. Carcinomatous and special forms of ulcer are taken up farther on.

While the *causation* of ulcer of the stomach is not clearly understood, there is no doubt that the coöperation of the gastric juice is the determining factor. A peptic ulcer develops only in those situations subjected to the action of this secretion, i. e., in the lower end of the esophagus, the stomach, the upper portion of the duodenum, and in the jejunum after gastro-enterostomy. That the gastric juice alone is not responsible for the ulcer is also true. Numerous cases of hyperacidity never develop ulcer; ulcers appear only when nourishment of the gastric wall is interfered with. This may be the outcome of circulatory disturbances, i. e., diseases of the blood vessels, arterial spasm, embolism and thrombosis; or it may follow the infliction of trauma to the gastric mucosa. That trauma alone is probably not responsible for ulcer is shown by the promptness with which surgical wounds of the stomach heal. It seems to have been shown that anemia (chlorosis) predisposes to ulcer of the stomach (Kausch[1]).

With regard to the causative relationship between ulcer, hyperacidity, motor disturbances, and pylorospasm, one is justified in ascribing the occurrence of the last three to ulcer, but never the reverse. Robson[2] regards most gastric ulcers as septic in origin and charges the teeth with the responsibility in this connection. The bactericidal action of gastric juice, the sparse bacterial flora in the stomach, and the resistance the normal mucosa possesses with regard to infection argue strongly against this theory. On the other hand, it is conceivable that hematogenous invasion of the blood vessels of the stomach

(bacterial embolism), occurring in connection with acute infectious diseases, may readily stand in a causative relationship to ulcer of the stomach, in which event the mechanism of its production in this class of cases is quite in accord with that already stated.

Gastric ulcer is *most frequently situated* in the posterior wall of the stomach, in the aboral end of the lesser curvature. Four fifths of all lesions are located in this restricted area. The ulcer varies in size from that of a pin head to a dinner plate. It is usually round or oval but may be irregular in outline. Occasionally the process encircles

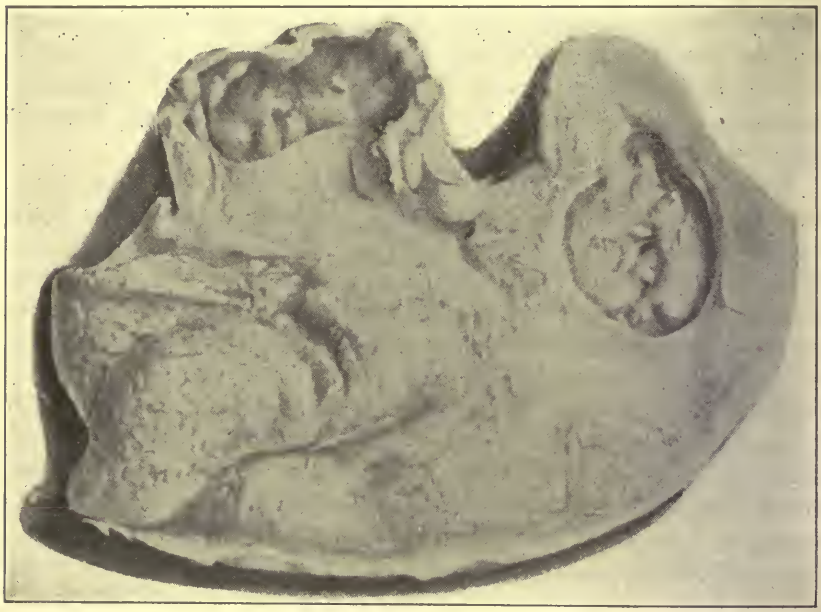

FIG. 908.— GASTRIC (PEPTIC) ULCER (from MacCallum's Text Book of Pathology).

the stomach in the form of a ring. The edge of the ulcer is usually sharply defined and elevated beyond the niveau of the surrounding mucosa. Old ulcers are indurated; as a rule the deeper portions are funnel shaped. The depth of the ulcer does not accord with its extent. As the process extends to the visceral peritoneum, the latter is either perforated or, which is more common, circumscribed peritonitis (perigastritis) develops and this results in adherence of the stomach to an adjacent part — the pancreas, liver, colon, and anterior abdominal wall in the order of frequency mentioned. The infrequency with

which an ulcer adheres to the anterior abdominal wall accounts for the fact that eighty per cent of the perforations occur upon the anterior surface of the stomach. Of these, three out of five are nearer the cardia than the pylorus. According to Moynihan,[3] twenty per cent of perforating ulcers are multiple. In a certain number of instances of perforation, the resultant local peritoneal inflammation recedes spontaneously, leaving a number of adhesions; in another class of cases the peritoneal exudate suppurates, and abscess is formed, which ruptures into the general peritoneal sac, with dire consequences.

Hemorrhage occurs in fifty per cent of gastric ulcers and is due to erosion into a blood vessel. In the other cases destruction of the vessel is preceded by thrombosis, which prevents the bleeding. Recurrent bleeding is the result of invasion of additional blood vessels.

Healing of the ulcer occurs in various ways. Superficial ulcers heal without perceptible scarring; deeper ones cause cicatricial deformation of the stomach wall. The scar of an ulcer may subsequently break down and a secondary ulcer form. There is no doubt that carcinoma develops at the site of an ulcer or a sequential scar.

Ulcer of the stomach presents an exceedingly varied *clinical picture.* A large ulcer may not produce symptoms, while a small lesion is often attended with distressing disturbances. The general condition of the host is unfavorably influenced by bleeding and the inability to absorb nourishment. Rapid and progressive deterioration of general health indicates a large ulcer or the presence of carcinoma. However, this may also occur in connection with neuroses. Strange to say, about one fourth of the cases of persistent ulcer die of *pulmonary tuberculosis.* All gastric disturbances are accompanied by hypochondria, neurasthenia, hysteria, etc. It is often impossible to determine which is primary.

Digestive disturbances, so-called dyspepsia, are not characteristic. They consist in loss of appetite, pressure sensations, eructations, and "heartburn." Constipation is the rule; in a general way, diarrhea argues against ulcer. *Pain* is a common symptom; it is characterized as cramp-like or burning, and is often provoked by the ingestion of food and by pressure over the epigastrium. Unfortunately, cholecystitis, gastritis, pancreatitis, and ulcer of the duodenum also give rise to pain in the gastric region. *Vomiting* is rarely absent.

Gastric hemorrhage is perhaps the only certain sign of gastric ulcer. It occurs, however, only in a small number of cases. The bleeding is divided into *severe* and *slight.* The former is fatal in about five per

cent of the cases; slight bleeding may persist for a long time and give rise to secondary anemia. *Hematemesis* occurs when the stomach is suddenly flooded with a large quantity of blood. The presence of occult blood should be sought for in all suspicious cases. While gastric hemorrhage is indicative of ulcer, it also occurs from the varicosities developed in connection with obstruction of the portal circulation, in hemophilia, and in simple erosion of blood vessels.

Ulcer is almost always attended with *motor disturbances* of the stomach as follows: 1. a pyloric ulcer may cause deformation of the stomach; 2. all ulcers provoke spasmodic contraction of the pyloric musculature; 3. adhesions at the pylorus cause kinking and obstruction at the aboral end of the stomach. However, ulcers situated in other sectors of the stomach frequently cause reflex pylorospasm.

Hyperacidity is usually present in open ulcer of the stomach. In some cases the hydrochloric acid content of the gastric juice is normal; it is below normal only in severe cases of anemia or complicating gastritis. Hyperacidity is also attendant upon benign pyloric stenosis and gastric neuroses. When carcinoma develops in a gastric ulcer, hyperacidity persists. All this means that hyperacidity possesses only relative diagnostic value. However, it is a substantial factor in the sum total of the symptoms.

The *Röntgenographic findings,* if positive, are of great significance but are not to be regarded as determining in a negative sense.

Occasionally the infiltrated area of a gastric ulcer is palpable through the abdominal wall. Palpation of a diffuse infiltrate suggests perigastritis. Circumscribed tumor indicates that the ulcerative process has become adherent to the anterior abdominal wall. In the latter event, the mass does not move with respiration.

Perforation into the general peritoneal sac is an important complication of gastric ulcer. As already stated, this occurs most frequently at the anterior aspect of the stomach. Clinically, perforating ulcer may be divided into two forms: in the one, the patient gives a history more or less indicative of ulcer; in the other, and this is not rare, the perforation occurs "out of a clear sky." The diagnosis is as easy in the first class of cases as it is difficult in the second. The clinical picture of perforating gastric ulcer has already been discussed in connection with peritonitis. At this time, it is perhaps permissible to remind the reader of the almost characteristic BOARD-LIKE RIGIDITY OF THE ABDOMINAL WALL AND THE SEVERITY OF THE SHOCK in this class of cases.

In the *differential diagnosis* it is necessary to consider that

1. Carcinoma must be taken into account, not only as a primary condition but also from the standpoint of its development in connection with ulcer or a cicatricial area consequent to it. Differentiation is based on the history, the age of the patient, his general condition, loss of weight, cachexia, the analysis of the gastric contents, the character of bleeding, and the physical examination. When operative measures of relief are indicated, the differential diagnosis is not so essential. However, the clinical manifestations must always be elucidated, so that they may aid in deciding the surgical procedure when the abdomen is opened.

2. In not a few instances the manifestations attributed to ulcer of the stomach are the outcome of lesions of the *gallbladder* and *biliary passages,* especially when the latter are attended with the formation of adhesions which cause disturbances of gastric digestion. In these cases a careful analysis of all the available factors must be taken into account and if this does not lead to a definite conclusion the surgeon must formulate the surgical attack accordingly.

3. *Epigastric hernia* gives rise, at times, to a high degree of gastric disturbances.

4. *Duodenal ulcer* must always be taken into account.

5. It occasionally happens that operative exposure of the stomach does not reveal a discernible lesion. In these cases the disturbances are attributable to *gastralgia, nervous dyspepsia* with hyperacidity, and primary *gastric atony.* While this misfortune is rare, it is also proper to add that it has occurred when due consideration has been given the problem in hand.

Robson[2] says "It may be ordinarily concluded that epigastric pain related to food, vomiting, and localized epigastric tenderness, constitutes a combination of symptoms pointing to ulcer, but that if hematemesis is superadded, the existence of ulcer is almost certain."

The *surgical treatment* may be directed against the ulcer or its complications. Originally, surgical efforts at relief were directed only toward combating the complications which threatened life. As surgical technic has improved, measures have been devised that not only overcome the complications but result in healing their cause.

The complications are discussed first:

1. *Acute perforation* imperatively demands celiotomy and repair of the lesion. The technic of the procedure has already been taken up (p. 1917).

The presence of a stenosing pyloric ulcer brings up the question of simultaneous gastro-enterostomy. The latter is justified if the general condition of the patient permits. The combined operation was first performed by Braun[4] (1883). In this connection the work of English,[5] Barker,[6] Robson,[7] Ward,[8] and Moynihan[9] is valuable.

When *gradual perforation* is attended with *perigastritis,* haste in the employment of operative measures is objectionable. In these cases it is best to await cessation of the process, when the problem may be treated as in chronic ulcer, unless an abscess is formed. In the latter event, simple drainage of the exudate (as in appendicular abscess) is the procedure of choice.

2. In *organic pyloric stenosis,* attended with severe motor insufficiency, operative relief is indicated. The question as to the character of the operation has already been taken up (p. 1977).

3. *Bleeding* may require operative attack for two reasons, a. in the event of severe uncontrollable hemorrhage, b. in frequently recurring bleeding of moderate degree which seriously reduces the patient's general condition (chronic anemia).

a. The surgical indication to expose the bleeding area and arrest the hemorrhage in the usual way is dimmed by the following considerations: the bleeding may cease spontaneously, even in apparently hopeless cases; the menacing general condition of the patient makes operative measures exceedingly hazardous, and the bleeding point is often difficult to locate and may escape search. In any event, prolonged manipulations must be avoided and, if the source of hemorrhage is not readily located, gastrojejunostomy may be made with the view of unburdening the stomach. Temporary tamponade of the stomach has proved a life saving measure in a number of cases. However, despite the difficulties of the surgical problem, it must be remembered that five per cent of the cases of ulcer of the stomach die as the result of hemorrhage.

b. In the second class of cases the picture is gradually developed and the internist has an opportunity to employ the available therapeutic measures. Progressing anemia indicates that surgical efforts at relief should not be postponed. The problem is then reduced to healing the ulcer. The available methods are those described in connection with simple ulcer.

4. *Adhesions* are subjected to operative procedures in two classes of cases:

a. Loose adhesions which destroy the motility of a sector of the

stomach and cause deformation of the organ. These are usually the result of chronic perigastritis and are regarded as being responsible for *gastralgia* in a certain number of cases. Separation of the adhesions (gastralysis) has been followed by relief of the symptoms (v. Hacker[10]).

b. Adherence of a penetrating ulcer to the abdominal wall gives rise to severe pain. Relief follows resection of the affected area and repair of the stomach wall by suture. Inflammatory coalescence of the stomach with neighboring organs may present a difficult surgical problem. The aim should be to separate the adhesions and resect the ulcerated area.

5. Excluding those in which operation is absolutely or relatively indicated, there remains a class of cases unattended with complications dangerous to life. Despite careful attention to diet, etc., a number of these suffer pain, are badly nourished, and though vomiting is not necessarily a marked symptom, only certain articles of diet are retained. There is no doubt that operative procedures make it possible for a large number of those thus afflicted to return to a practically normal existence.

As to the method of procedure, *gastro-enterostomy* may be regarded as the operation of choice in uncomplicated gastric ulcer. The operation is followed by disappearance of the disturbances in motility; pain, and tenderness, and hyperacidity diminish. The fact that gastric ulcers heal after gastro-enterostomy is shown clinically and by autopsy. Kausch[1] says "I have on several occasions made a gastro-enterostomy for the relief of indurated ulcer and later opened the abdomen for the purpose of resection, and have been astonished to find the ulcer healed."

The palliative operation has, however, not gratified the ambitions of all surgeons, both as regards subsequent symptomatology and because of the disturbing consideration that a large number of undoubted ulcers later develop carcinoma. This has led to the more recent practice of resection of the ulcer. As, however, the V-shaped resection of a sector of the stomach is followed by disturbances in the innervation of the organ, *sectional resection and gastrogastrostomy* is employed in cases of indurated ulcer (Riedel and Payr,[11] Stewart[12]). The method has a not inconsiderable mortality and should be reserved for special cases, but is probably preferable to simple excision of the ulcer.

Pyloric exclusion in conjunction with gastro-enterostomy is indicated in ulcers located at the pylorus.

It is perhaps permissible to summarize the treatment of gastric ulcer as follows: FRESH UNCOMPLICATED ULCER BELONGS TO THE INTERNIST; GASTRIC BLEEDING ALSO BELONGS TO THE INTERNIST WITH THE SURGEON AT HIS ELBOW. COMPLICATED ULCERS BELONG TO THE SURGEON; CHRONIC UNCOMPLICATED ULCER SHOULD BE SUBJECTED TO OPERATION, IF THE SOCIAL CONDITION OF THE PATIENT DOES NOT PERMIT OF PROLONGED SEMI-INVALIDISM.

Ulcers of the Duodenum.— Peptic ulcer frequently appears in the duodenum. It does not occur in the rest of the gut.

The etiology, anatomy, and course of duodenal ulcer are similar to those located in the stomach. An interesting peculiarity of the lesion is its development in connection with *extensive freezing or burning of the surface.* In these instances the ulcer runs an acute course, is attended with more or less bleeding, and often perforates (according to Collins,[13] 60 per cent), usually into the general abdominal cavity.

The *clinical picture* of duodenal ulcer is very much the same as that of the gastric lesion. In typical cases the disturbances do not begin until two to four hours after ingestion of food. *Hunger pain,* relieved by bland articles of diet, is common. Pain and tenderness are located toward the right hypochondrium — the former radiates deeply. Periodical pain occurs in four fifths of the cases (Kemp[14]). Vomiting is unusual and is ascribable to stenosis.

Bleeding occurs in only seventeen per cent of the cases (Perry[15]) and is likely to be severe. The ulceration erodes the pancreatico-duodenal, the gastro-epiploica dextra, the pancreatic and the hepatic arteries. Blood may often be found in the stool.

Hyperacidity is as frequent as in gastric ulcer. Motor disturbances, especially pylorospasm, are the same as in the latter lesion. *Icterus,* which is not rare, is due to cicatricial stenosis of the papilla and to duodenitis.

Perforation presents no clearly defined distinctive features, except that the inception of the symptom complex is more marked on the right side. The peritoneal involvement is very likely to be mistaken for appendicitis. In a not inconsiderable number of cases, the exuded duodenal contents collects in the gutter between the transverse colon and the liver; the resultant adhesions divert the fluid along the ascending colon into the right iliac fossa and on into the pelvis; from there it travels along the sigmoid and descending colon, thus giving rise to an exceedingly complex clinical picture. In another class of cases, a posterior perforation occurs extraperitoneally; in these instances a

retroperitoneal phlegmon or a subphrenic abscess results (Lennander[16]).

Finally, duodenal ulcer is not rarely the site of *carcinomatous changes* and, in some instances, cicatricial contraction is followed by obstruction of the portal circulation (Moynihan[17]).

The *diagnosis* of duodenal ulcer is based more upon the history of the case than upon the objective findings. Of the former, food pain, hunger pain, and the periodicity of the attacks are indicative. The British and our own surgeons rely mostly upon the history of the case, although recently the latter have given due consideration to the refinements of the gastro-enterological examination (Kemp[14]). The *Röntgenographic* findings, if carefully interpreted, are of great diagnostic value and are becoming more so.

The grave *complications* coincident with duodenal ulcer render the affection a far more serious one than is the gastric lesion. Of 70 cases autopsied by Perry,[15] 20 died as the direct result of the ulcer; only 8 had healed. Many pathologists deny that spontaneous healing ever occurs.

The *surgical treatment* of duodenal ulcer until recently consisted in the operative relief of its complications. Our own surgeons, notably the Mayos,[18] have taught the world that operative measures may be successfully addressed to the ulcer, so that the complications may be avoided. While, in the hands of the especially skilled, resection of the ulcer is often successful, the procedure of choice would seem to consist in *gastrojejunostomy,* together with *exclusion of the pylorus.* The latter is best accomplished by division of the first portion of the duodenum, and closure of both ends by suture.

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CHAPTER VIII

TUMORS OF THE STOMACH

Benign Tumors.— Tumors of the stomach, other than carcinoma, are rare. Fibromata, fibrolipomata, myomata, and lymphadenomata develop primarily from the connective tissue. They possess more pathological than surgical interest. Occasionally a benign tumor of the pylorus produces symptoms of obstruction. Of this class of tumors *fibromata* are the most frequent. Allessandri[1] has collected thirty-two cases. Ebstein[2] analyzes twenty-four cases of adenoma.

Sarcomata.— Sarcomata are not so rare. According to Fenwick,[3] they comprise 5 to 8 per cent of all primary tumors of the stomach. The growth originates in the connective tissue of the submucosa and extends very rapidly. Lymphosarcoma and the round cell variety infiltrate the stomach wall; the spindle cell and mixed tumors are likely to be circumscribed. The *diagnosis* is not made before operation, and even after exposure may not be certain until microscopical examination clears up the problem (Cantwell[4]).

Carcinoma of the Stomach.— The only known etiological factor in carcinoma of the stomach is chronic ulcer. The pathological anatomy of the disease and the general considerations are taken up elsewhere in this work (Part VI, chap. xvii).

Primary carcinoma of the stomach may be of the cylindrical, spheroid or, very rarely, of the squamous type (Rolleston[5]). According to Dowd, 9,000 deaths occurred in the United States in 1900 from cancer of the stomach. The growth is most often located at the pylorus (60 per cent).

Cancer extends in the continuity of the stomach, mainly in the lymphatic spaces and vessels. In this way the process has a tendency to progress toward the lesser curvature. It is usually arrested at the pylorus but passes beyond it to a slight extent (½ to 1 cm.) in 31 per cent of the cases. In the direction of the cardia cancer takes its unrestricted way and, as the microscope shows, invades the tissues of the stomach wall beyond the grossly visible infiltrate. This is of

surgical importance — at least 5 to 10 cm. of the oral end of the stomach must be removed with the growth.

Outside of the wall of the stomach cancer spreads by way of the lymphatics (Cuneo[6]), all of which ultimately drain into the glands situated at the upper end of the pancreas behind the stomach. The glands are grouped around the origin of the celiac axis, spread out to the right on the pancreas as far as the pylorus, and to the left as far as the hilus of the spleen, and they also receive the afferent lymph flow from the liver, pancreas, and spleen, to empty finally into the thoracic duct. However, before the lymph vessels of the stomach reach the glands located at the upper border of the pancreas, they pass through a number of smaller glands situated along both curvatures of the stomach. The latter are arranged in three groups (see Fig. 885).

1. From by far the greatest extent of the stomach, the larger branches extend to the *lesser curvature and the cardia.* Thus, all the glands lying in front of, laterally to, and behind, the cardia and along the coronary artery (left) form a group called the *superior gastric.*

2. A few small elongated glands are found in the *right half of the greater curvature* and at the pylorus itself; these lie in the course of the gastro-epiploic artery.

3. The smallest lymphatic zone which corresponds to the *fundus half of the greater curvature* sends a few of its branches to the hilus of the spleen.

The regional lymph nodes are involved very early in carcinoma of the stomach. The invaded group corresponds to the location of the growth. The course of the lymph stream brings about invasion of the glands of the lesser curvature when the tumor is situated at the pylorus. The removal of these glands does not present technical difficulties. Removal of the glands located at the pancreas is almost impossible. Their involvement excludes the possibility of the radical operation.

The extent of glandular involvement is not proportionate to the size of the cancer but is closely related to the degree of its malignancy. Not all swollen glands are carcinomatous.

Hematogenous dissemination of cancer occurs by the veins of the stomach and the portal system. Direct extension invades the contiguous organs (liver, pancreas, and spleen).

Peritoneal metastases occur when the growth penetrates the stomach wall. These are likely to be widely disseminated throughout the entire abdominal cavity.

Ulceration occurs early in cancer of the stomach. It must be differentiated from carcinomatous ulcer. This is not always easy. The ulcerative process soon erodes the walls of the blood vessels, thus giving rise to more or less bleeding. Perforation and peritonitis are not uncommon.

The *symptoms* of carcinoma of the stomach are due in part to the cancer itself, and in part to the functional disturbances it causes, depending upon the particular part of the stomach invaded by the growth.

The General Condition of the Patient.— When the cancer is located near the cardia or pylorus, the mechanical interference with nutrition soon leads to emaciation and hydremia. Unobstructing cancer affects the general health of the host very gradually and at first only by disturbing the digestive function. Cachexia develops late, but is preceded by increasing anemia; the hemoglobin count is likely to fall to 30 per cent and lower. In all cases a search for lymphatic and general organ metastases should be made. The latter may first attract the attention of the patient. Constipation is the rule.

Motility.— As in ulcer, a stenosing lesion must be differentiated from a non-stenosing. However, the latter also produces disturbances of gastric motility secondary to damage to the stomach wall.

Acidity.— Carcinoma is attended early in its progress with catarrh and atrophy of the stomach wall, so that the quantity of secreted acid and ferments is soon reduced. This is characteristic of carcinoma. The bacterial flora increases rapidly and lactic acid fermentation is increased. On the other hand, the hyperacidity persists when carcinoma develops in an ulcer. The appearance of lactic acid is indicative of carcinoma. Unfortunately this occurs late in the process. When, in the presence of lactic acid fermentation, the tumor is not palpable, the carcinoma is probably situated in the posterior wall of the stomach. *Blood* always appears in the vomitus and in the stool. Severe hemorrhage is not common.

A *palpable tumor* is an important diagnostic sign. Its dimensions and form are exceedingly variable. Small tumors of the pylorus are usually circumscribed; extension of the process along the lesser curvature is frequently palpable in the form of a crescent, its concavity pointing to the left. Growths of the lesser curvature alone present the outline just described, minus the pyloric mass. Cancers beginning at the cardia and at the fundus are not palpable until late in the process, when they appear as hard masses at the left border of the ribs. The

surface of the tumor is usually smooth at first, but soon becomes nodular. The latter is due to lymphatic involvement.

Non-stenosing carcinoma of the stomach, in itself, presents the picture of cancer. As a rule the primary lesion is located at the lesser curvature; occasionally, at the fundus. However, cancer of the cardia or of the pylorus may advance to a considerable extent without causing obstruction. In non-stenosing pyloric cancer the symptoms of mechanical obstruction are due to muscular insufficiency. However, this occurs late in the disease.

In the *differential diagnosis,* when a tumor is not palpable, chronic gastritis, atrophy, and chronic ulcer must be taken into account. The last is especially difficult to differentiate when a small mass is palpable and when the process is attended with severe anemia and secondary gastric atrophy. In addition to this, the presence of a tumor involves the question of the location of the lesion in organs contiguous to the stomach, i. e., the pancreas, liver, colon, and omentum.

Carcinoma of the cardia presents the clinical picture of esophageal stenosis. These symptoms are not likely to appear until the lesion is advanced. Unless the disease obstructs, it may escape recognition for a long time; on the other hand, the esophagoscope reveals the condition at once.

Cancer of the exit of the stomach is attended with the picture of pyloric stenosis and mechanical gastric insufficiency. Vomiting of fermented stomach contents occurs early and presents the characteristics already taken up. Reduction in the general condition of the patient is rapid. The picture of inanition precedes that of anemia and cachexia.

The *differential diagnosis* takes into account pyloric obstruction from other causes (ulcer, kinking, perigastritis, external pressure) and atonic insufficiency. In this connection chemical and microscopical examination of the stomach contents is very important. Ulcer-carcinoma, with hydrochloric acid and no lactic acid, cannot be differentiated from ulcer.

Easy as the *diagnosis* of advanced carcinoma of the stomach is, its early recognition is correspondingly difficult. Despite the employment of the available diagnostic methods, the problem often remains unsolved. Of these, the present day method of riadography is perhaps the most useful. This brings up the question of *exploratory celiotomy.* In this connection Robson[7] says "Whenever a patient, at or near middle life, complains suddenly of gastric uneasiness, pain

and vomiting, followed by progressive loss of weight and energy, and this is associated with anemia, the possibility of cancer of the stomach should be recognized; and in a suspected case, if no improvement takes place in a few weeks at most, and if repeated examinations of the stomach contents after test meals show diminished digestive power with a diminution or absence of HCl. and the presence of lactic acid, an exploratory operation is more than justified."

The *treatment of cancer of the stomach* consists in radical excision as long as this is attainable. Only the presence of metastases and severe cachexia are contra-indications in this connection. Neither immobility of the tumor nor its size precludes the possibility of a successful operation. The surgeon must not permit technical difficulties to restrict his efforts. The decision as to the inoperability of the process is often, to no slight degree, a question of the surgeon's realization of the exact condition when the problem is uncovered by celiotomy. In these instances he is called upon to decide an exceedingly important question, and to formulate the procedure accordingly. In all cases the character of the process must be decided first, i. e., whether it is carcinomatous or not. The most difficult problem, of course, arises in connection with ulcer-carcinoma.

The *size and extent of the tumor* in the wall of the stomach does not preclude extirpation. The entire stomach has been removed and the esophagus united to the jejunum. Although this is technically feasible, it is difficult to believe that such extensive involvement is not attended with lymphatic metastatic invasion making the resection useless as a therapeutic measure.

Extension of the process to the *transverse colon* calls for simultaneous resection of the latter. However, this step greatly increases the operative mortality. Direct extension of the tumor to the liver need not exclude gastrectomy, but the measure only prolongs life and makes it more bearable. On the other hand, adherence of the stomach to the *liver*, the result of perigastritis following ulcer, does not militate against the operation. Coalescence of the growth with the *pancreas* increases the technical difficulties very much. Small sectors of the pancreas may be excised; the removal of larger ones is followed by grave disturbances. The *spleen* is rarely involved in cancer of the stomach. Its removal is permissible.

In almost all cases of cancer of the stomach the *lymphatic glands* are found more or less involved in the process. Invasion of the glands located at the curvatures does not preclude radical extirpation of the

disease. However, when the glands in the lesser omentum, as far as is discernible by the palpating hand, are invaded, the radical operation does not serve a useful purpose. The pancreatic glands are particularly discouraging in this connection; their moderate involvement precludes removal of the process when a similar degree of invasion of glands in other situations is technically easy and purposeful. Extension of the disease to the glands situated at the portal vein and behind the peritoneum constitutes an insurmountable difficulty.

Metastatic deposits in the liver and peritoneum preclude the possibility of affording relief. Resection as a palliative measure is not justified.

The resection is carried out in the manner already described (p. 1930. The surgeon is not likely to err on the side of radicalism.

When resection is contra-indicated, the question of pallative operative measures comes up. These consist in *gastro-enterostomy, gastrostomy, pyloric exclusion, and jejunostomy.*

Gastro-enterostomy is permissible in the event of severe motor disturbances, when cachexia has not advanced too far.

Gastrostomy is a justifiable measure of relief in obstructive carcinoma of the cardia. *Jejunostomy* is rarely executed. *Pyloric exclusion* is of doubtful usefulness in this class of cases.

The *results* of gastrectomy justify employment of the measure. As the technical problem presented by the various cases cannot be taken into account, a mortality rate based on statistics has no value. This aspect of the situation is discussed by Robson,[7] Moynihan,[8] and Kausch.[9]

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CHAPTER IX

SURGERY OF THE INTESTINES

Congenital Anomalies of the Gut.— Of the congenital anomalies of the gut, *Meckel's diverticulum* is the most frequent; it consists of a form of incomplete obliteration of the omphalo-enteric duct. According to the statistics of the English Anatomical Society, the duct remains patent in two per cent of all individuals. The diverticulum is a blind sacculated protrusion originating from the lower portion of the ileum (about 60 to 70 cm. above the valve of Bauhin) at the free border of the gut. It may end in a pouch, free in the abdomen, or may be connected with the navel by a cord. Its size varies from a barely perceptible elevation to a tubular process thirty centimeters in length, which may be one centimeter in diameter or as wide as the ileum itself.

The *clinical importance* of Meckel's diverticulum lies in the complications developing in connection with it. Of these, the following possess surgical importance: (1) The divertieulum gains access to a hernia sac and resembles an inearcerated hernia. (2) It may be responsible for ileus, by snaring or by strangulation of gut forced into it. The diverticulum may become invaginated into the gut or *vice versa.* (3) It may develop into an extensive retention cyst. (4) Foreign bodies may lodge in its lumen. (5) The diverticulum may become the seat of inflammatory lesions; these present the clinical picture of appendicitis, and are not readily diagnosticated from the latter.

When celiotomy demonstrates the seat of an affection involving Meckel's diverticulum, it is promptly resected. If the anomaly is met with in the course of an abdominal operation, its tendency to produce disturbances justifies its removal. The technic of the procedure is similar to that described in connection with appendectomy (p. 1956).

Congenital stenoses of the gut are not rare. They have been observed in all sectors of the gut, including the anus, but most frequently occur in the rectum and at the anus; next to this they are

found most often at the site of origin of the omphalo-enteric duct. The duodenum, just above the papilla, is not infrequently the site of this class of lesion.

The *clinical picture* of intestinal stenosis is that of acquired chronic obstruction (p. 1996). The *treatment* aims at removal of the obstruction. The technic of the various procedures is taken up in connection with the surgery of the different sectors of the intestinal tract.

Congenital dilatation and hypertrophy of the *sigmoid flexure* (Hirschsprung's disease), *megacolon congenitum,* deserves special consideration. It appears to be due to a valvular obstruction which is not demonstrable in the excised specimen. The valvular mechanism, which acts only in an aboral sense, becomes increasingly operative as the gut is filled. The valvular closure is located at the sigmoid-rectal junction (Perthes[1]). The prolonged battle over the question as to whether the dilatation or the valvular obstruction is primary seems to have ended in favor of the latter by the investigations of Konjetzny.[2] The rest of the colon is also frequently dilated.

The disturbances consequent to the abnormality begin in childhood, although the host often drags himself into adult life. The *symptoms* are those of chronic intestinel obstruction, soon to be discussed (p. 000). The characteristic sign is that, when a large quantity of fluid is injected into the lower bowel, only the portion retained in the rectum returns, until a tube is inserted into the sigmoid, when the rest of the fluid is suddenly expelled. The dilatation may fill the greater part of the abdomen and cause it to be enormously distended.

The *treatment* consists in resection of the entire flexure, together with the valve. The operation is best made in two sittings.

Of the other congenital anomalies the following deserve mention, *abnormal length, or abnormal shortness, or absence of certain sectors* of the gut. These are at times responsible for volvulus.

The *cecum mobile* is regarded by some surgeons as responsible for atony, chronic obstipation, and chronic appendicitis. In these cases removal of the appendix does not relieve the symptoms. The surgeons who regard the condition as a clinical entity fix the cecum by plicating, by exclusion, or by resection. The rationale of these procedures is not proved.

General Considerations Regarding Chronic Intestinal Stenosis.—Before farther discussion of diseases of the gut, it is advantageous to take under consideration a symptom complex appearing in connection with affections of the intestines (inflammatory processes,

ulcers, scars, tumors, etc.), which, irrespective of the underlying cause, is attended with the same clinical picture. This symptom complex is always the outcome of lessening of the intestinal lumen, although the causative lesion gives little or no evidence of its presence. That is to say, the clinical picture of chronic intestinal stenosis dominates the situation so much that its cause cannot be established. In this regard the condition is analogous to acute ileus.

As a matter of fact, ileus is also a symptom complex produced by a number of changes in the gut. When either of these conditions is looked upon as a clinical entity, this is because we are unable to diagnosticate the underlying causative factor. Fortunately, this does not rob the surgeon of the therapeutic indication, although it is often responsible for delay in application of surgical measures of relief.

The term *intestinal stenosis* has a variable significance, depending upon its anatomical or clinical sense. From an anatomical standpoint, the slightest reduction in the lumen of the gut is a stenosis; from the clinical, only those which disturb the passage of the intestinal contents are considered as such. A considerable lessening of the lumen of the gut is necessary before propulsion of the contents is seriously interfered with, and so it follows that little additional increase in the obstruction is required to establish the picture of ileus. In this way one symptom complex merges into the other, or varies with the other, according to whether the stenosing process is progressive (tumor) and leads to a complete occlusion, or a primarily complete obstruction gradually merges into a simple narrowing (invagination). A stenosing lesion taking its insidious chronic course may fulminate into the picture of acute intestinal obstruction, if a foreign body, or, in the colon, if a hard portion of feces is incarcerated in the narrowed area, or if a sudden muscular insufficiency of the oral gut occurs.

The lesions responsible for chronic intestinal stenosis are those developing in the gut itself and those acting from without. The former are scars following ulcerative processes, tumors, foreign bodies, intussusception, and spasm; the latter are tumors that make pressure, abscesses, kinking, and peritoneal adhesions.

The *consequences* of a stenosing lesion depend upon the effect it produces upon the progress of the intestinal contents, and this varies with the character of the latter. Fluid feces are propelled through a markedly lessened lumen, where the more concentrated are likely to be arrested. When the intestinal flow is hindered, the intestine undergoes certain *anatomical changes*. The oral gut becomes distended and

its musculature attempts to overcome the obstruction by increased peristalsis. In a few days the musculature begins to hypertrophy and this may be compensatory in character for a long period of time (years). The hypertrophy is genuine (not a hyperplasia), but has its limitation, so that if the hindrance persists or becomes greater (which in chronic stenosis is the rule), the oral gut dilates and ultimately becomes paralyzed. These changes are very similar to those occurring in other hollow viscera — the heart, the bladder, etc. As distention increases, the circulation in the gut is impaired, at times to such an extent that perforation occurs. The lumen of the aboral portion of the gut is reduced commensurate with the degree of stenosis.

The *clinical manifestations* of intestinal stenosis vary with its location, the length of the oral sector of gut, and the contents of the latter. A division in this connection may be made into *duodenal, small and large intestinal,* and *rectal stenoses.* The last are taken up in Part, XVII, chap. v.

The picture of *duodenal stenosis* varies in accord with whether the obstruction is located above or below the opening of the bile and pancreatic ducts.

Suprapapillary stenosis resembles pyloric obstruction; it causes not alone obstruction of the upper portion of the duodenum, but is also responsible for pyloric insufficiency and dilatation of the stomach. Its differentiation from pyloric obstruction is possible only by the Röntgenographic findings. The secretions of the upper duodenal sector do not impart any peculiarities to the vomitus.

In *infrapapillary stenosis* the biliary and pancreatic secretions collect in the duodenum and flow backward into the stomach. When the stomach is emptied by lavage and syphonage, the clear fluid return is suddenly, without retching on the part of the patient, stained with bile; this is characteristic. If the stenosis is complete, the intestinal contents give evidence of the absence of biliary and pancreatic secretions.

Papillary stenosis presents a characteristic picture, especially in cases of carcinoma of the duodenum. The lesion is attended with slowly increasing icterus. The stool is devoid of bile. The interference with pancreatic secretion is shown by the presence of fat in the stool, and, at times, by glycosuria.

The disturbances consequent to *stenosis of the upper jejunum* are similar to those occurring in connection with infrapapillary lesions. The lower the stenosis is located in the small intestine, the less marked

are the gastric symptoms and the more dominant the intestinal. Distinct gastric symptoms are present only when the obstruction is of a high degree. Periodical vomiting indicates temporary complete obstruction of the gut and subsequent reëstablishment of the lumen; vomiting may also be reflex, especially in cases of lesions in the colon. In all cases of intestinal stenosis the patient complains of digestive disturbances, the tongue is coated, the appetite poor.

The most important symptoms in intestinal stenosis are *visible and palpable distention* and *increased peristalsis* above the site of the stenosis. If the stenosis is located high in the gut, the distention is slight, as the arrested gases and fluid readily gain access to the stomach. The degree of local meteorism is greater, the lower the stenosis is situated in the gut.

The distention is at times diffuse; at others, local; not infrequently the contour of the gut is readily visible. This depends upon the thinness of the abdominal wall, the outline of the gut not being visible in the normal abdomen. In most instances the surgeon is dealing with increased peristalsis of a restricted sector of the gut, which is usually intermittent in character, but may cause fixation of the gut in its dilated state (*intestinal rigidity*).

Pain in intestinal stenosis is caused by the forced peristalsis, and is colicky in character.

Intestinal rigidity is the most important symptom of chronic stenosis. It is differentiated from the increased peristalsis of nervous origin, in that the latter is general while the former is localized. A superficially located intestinal loop emits a high pitched percussion note, and at times metallic tinkling may be elicited.

Stenosis of the small gut has no influence on the *form of the stool*, but its quantity is in accord with the degree of obstruction. Constipation is the rule; intermittent diarrhea occurs when enteritis coëxists. The urine is always scanty.

Stenosis of the colon is attended with disturbances which vary with the proximity of the lesion to the ileocecal valve. Obstruction of the colon is attended with a lesser degree of increased peristalsis than obtains with the small gut; that of the latter is twenty-five times more active than the former. A mass may repose indolently in the colon for days without causing disturbances. Only a complete occlusion of the colon provokes stormy manifestations. Carcinoma of the sigmoid may remain quiescent until almost complete obstruction occurs.

Stenoses located at the cecum and ascending colon produce a clinical

picture similar to that coincident to an occluding lesion of the ileum. The differential diagnosis is based on evidence of distention of the cecum; this is usually easily recognized.

Differentiation between stenoses of the upper and lower sectors of the colon is based on the primary seat of the distention — i. e., whether it is located to the right or the left of the vertebral column. In stenosis of the rectum or of the sigmoid flexure, both iliac fossae are distended; when the obstruction is higher, only the right is distended. The lower the occlusion, the more is defecation influenced; diarrhea alternates with obstipation. Diarrhea indicates secondary colitis. In other respects the stool is not characteristic. Deformation of the expelled stool (band, ribbon, or pencil shaped) occurs only when the stenosis is located low in the sigmoid or in the rectum. On the other hand, the presence of pus, blood or fragments of a tumor in the ejecta are of diagnostic significance. Valuable information is obtained with the help of the *sigmoidoscope*.

The effect of the stenosis upon the general health of the patient is mostly dependent upon the character of the lesion. In a general way, one is justified in stating that all stenoses involve grave menace to life. The chief danger consists in complete obstruction and distention perforation.

The *treatment of chronic intestinal stenosis* consists in the employment of surgical measures as follows:

1. Complete extirpation of the stenosis by resection is permissible only in cases of malignant disease; in tuberculous and other ulcerative processes, the indication is relative.
2. Simple entero-anastomsis.
3. Complete or incomplete intestinal exclusion.
4. Anus preternaturalis. See Operations upon the Intestines, chap. iii, of this part.

Ulcers of the Small and Large Gut and Their Complications.— Numerous forms of ulcer appear in the small and large intestine; most of these do not possess surgical importance. Ulcerative processes located in the colon (colitis ulcerosa), chronic dysentery, and tuberculosis are at times subjected to surgical measures of relief. The operation, in the milder cases, consists in enterectomy; in the more severe, the establishment of an artificial anus is justified. Ileosigmoidostomy and intestinal occlusion have not given satisfactory results.

It is at this time permissible to allude to the recent treatment of mucous colitis and chronic obstipation by operative measures (Lane[3]).

The operations consist in ileosigmoidostomy, cecotransversectomy, ileotransversectomy, unilateral colonic exclusion, colostomy, etc. When the nervous origin of the affections stated is taken into account, it is not surprising that the ultimate outcome of the measures has been disappointing. In a not inconsiderable number of instances, obstipation has been succeeded by diarrhea.

Other than this, the complications of intestinal ulceration demand surgical intervention; namely, *bleeding, perforation, stenosis* and *tumor.*

Bleeding does not occupy a prominent position in intestinal surgery. It occurs most frequently in connection with typhoid fever, but also in ulcers of other forms. The difficulty in locating the source of the hemorrhage makes its exposure and its arrest almost impossible.

Perforation of an intestinal ulcer in connection with lues and tuberculosis is rare. On the other hand, typhoid perforation is a frequent occurrence (see statistics of Harte and Ashurst[4]). In two thirds of the cases the perforation is located in the lower ileum; of these, fifteen per cent are multiple.

The *clinical picture and the diagnosis* of ulcerative perforation of the gut are similar to that discussed in connection with the traumatic lesion. The perforation leads to the various forms of peritonitis, also already described (p. 1890). The *diagnosis* may be difficult when the causative ulcer has not given rise to symptoms; on the other hand, it does not present difficulties when the perforation occurs in connection with typhoid fever. The sudden appearance of shock, followed by severe pain and the evidences of peritoneal irritation, leaves little room for doubt.

The *treatment* of perforated ulcer of the gut has already been taken up in connection with abdominal operations (chap. iii).

Stenosis of the Gut.— Every deeply extending ulcer of the gut is likely to produce stenosis of its lumen. Of these, the tuberculous is the most common.

Given a case of intestinal stenosis, one is justified in assuming its *tuberculous* origin unless another cause is clearly manifest.

The assumption that tuberculosis of the intestine is a fatal affliction is erroneous; this is true only when there is a coëxisting advanced pulmonary tuberculosis, and in the miliary form of the disease. Of one thousand cases of tuberculosis autopsied by Eisenhardt,[5] 563 showed gastrointestinal tuberculosis. Of thirty-five cases of completely

or partially healed ulceration, nine caused stenoses of the gut. The ileocecal region is most often involved.

In most cases coming under the observation of the surgeon, the ulcer is healed and the establishment of its character is not easy. The form of the stenosis varies from a small scar to complete tubular narrowing of the lumen. In many instances, multiple stenoses are present. The solitary strictures are usually located at the cecum. The oral portion of the gut, obstructed by a tuberculous lesion, undergoes the typical changes already described. It is not possible to *diagnosticate* the tuberculous nature of the process clinically, although the gradual development of the picture of intestinal stenosis in a tuberculous patient permits of the assumption that the lesion is tuberculous in character.

The surgical *treatment* of tuberculous stricture, when the process is healed, consists in entero-anastomosis. When the process is still active, resection of the involved area is the method of choice. The possibility of luetic stricture must not be lost sight of.

Tumor forming tuberculous ulceration, occurs most frequently in the ilocecal region (tuberculous ileocecal tumor). Borchard[6] reports a case of syphiloma of the sigmoid which healed after the establishment of a colostomy opening and the use of antiluetic treatment. However, tuberculous tumors have been found in this situation and also in other portions of the colon and in the small gut. *Actinomycosis* develops in the same situations, and presents a picture very similar to that of tuberculosis.

The peculiarities of the process consist in a chronic inflammatory thickening of a localized sector of gut, in contradistinction to the thinning out attendant upon the disseminated form of the affection.

Two forms of tumorous intestinal tuberculosis are described (Conrath[7]). The first, the most frequent, originates in the serosa in the form of tuberculous inflammation of the peritoneum covering the ileocecum, and no doubt is lymphogenous. At first, the lesion consists in a number of minute tubercles, which become confluent; this it attended with massive connective tissue hyperplasia. The musculature of the gut acts as a barrier for a long time, but ultimately is infiltrated by the persistent invader, which finally, though very late in the disease, perforates, gains access to the mucosa, and extends there in the form of a tuberculous ulcer. The second form of the disease begins as tuberculosis of the mucosa of the ileocecal region. Why at one time tuberculosis, which shows a tendency to heal, should form a stricture, and at another should be attended with massive tumor

formation, is not known. The second form of tuberculosis develops in the submucosa and in all other respects takes the same course as that which originates in the peritoneum.

The disease is essentially one of early life (between twenty and forty), and usually occurs in pale, phthisical looking persons; it is often preceded by the clinical picture of a low grade of appendicitis; in other instances, it presents the symptoms of gradually increasing intestinal stenosis. Diarrhea is very rare. The first indication is the appearance of a tumor.

Tuberculous ileocecal tumors are hard, nodular, and often visible through the abdominal wall in the form of a sausage shaped mass, the outline of which conforms in a general way to that of the cecum and ascending colon. It can rarely be grasped in the hand and usually is not painful. In other respects the tumor possesses the characteristics of an ileocecal growth; it does not move with respiration, and is dull on percussion. After a time the mass becomes fixed, as the lymphatic nodes of the mesentery and mesocolon are invaded and the process extends to contiguous parts and organs. In a certain number of instances, a cold abscess is formed; this may perforate into the gut, the bladder, the rectum, the pleura, etc.

The disease follows an essentially chronic course and shows little tendency to heal spontaneously. Unless surgical measures of relief are employed, the outcome is fatal; the patient dies of intestinal obstruction or from tuberculosis of other organs.

The *differential diagnosis* takes into account other tumors appearing in this region — not always an easy problem; even the microscopical examination is not determining in every instance.

The *therapy of ileocecal tuberculosis* wavers between exclusion of the diseased area by entero-anastomosis and resection. The former is never followed by complete relief, but may be necessary because of technical difficulties. Resection and subsequent ileocolostomy (p. 1949) is the operation of choice and is followed by brilliant results. In cases in which the general condition of the patient does not warrant extensive surgical manipulations, the three stage operation (p. 1954) may be utilized.

Acquired intestinal diverticula may make their appearance in the guise of tumors. Acquired diverticula, *classified on an etiological basis*, are either pressure or traction protrusions. The former are caused by ulcers which lessen the resistance of the intestinal wall, or by foreign bodies and gallstones, with or without ulceration. Traction di-

verticula develop as the result of adhesions and tumors which drag out a sector of the wall of the gut.

Diverticula are usually located in portions of the gut not covered by serosa, and, therefore possess lessened resistance, i. e., where the vessels extend to the intestine between the layers of the mesentery. For this reason the duodenum is most often affected. Occasionally a solitary diverticulum, which may reach great size, develops at the sigmoid flexure. Although its exact cause is unknown, it is probably congenital in origin.

The chief *clinical significance* of the acquired diverticula lies in their resemblance to tumors. The process usually is not recognized until it is exposed by celiotomy. The diverticula should be extirpated.

Benign Tumors of the Intestines.— Benign tumors of the gut are rare. *Adenomata, lipomata, fibromata, myxomata, myomata, angiomata, and mixed tumors* have all occurred in this situation.

Adenomata usually arise from the follicles of Lieberkühn and are flat or pedunculated, single or multiple; they vary in size from that of a pea to the adult fist. They occur early in life and, while they may develop at any portion of the gut, are most often found in the *rectum*. A special form of multiple polypoid adenomata involves large areas of the intestinal mucosa and is often seen in several members of the same family. As a rule, the rectum and colon are invaded; the tumors show a tendency to become malignant.

Only the *complications* of benign tumors possess surgical importance. The tumor may cause *intussusception* provoked by the peristalsis, which telescopes the gut as it tries to expell the growth; or the process may occlude the lumen and give rise to *obstruction*.

The *treatment* consists in correction of the intussusception and extirpation of the tumor by enterotomy or enterectomy.

Sarcoma of the gut, compared to carcinoma, is rare, and, in contradistinction to the latter, involves the small intestine more often than the large. Strange to say, sarcoma, especially lymphosarcoma, seems to develop most frequently in connection with tuberculosis of the intestine. That this indicates a relationship between chronic inflammatory processes and connective tissue new growths has suggested itself to not a few pathologists.

Clinically, sarcoma of the gut (as elsewhere) is characterized by its rapid growth, often *without the evidences of intestinal stenosis*. On the contrary, the gut at the site of the lesion is likely to be dilated (Baltzer[8]). The *diagnosis* will, of course, only be presumptive. The

treatment, i. e., extirpation of the growth, is not followed by satisfactory results.

Carcinoma of the Intestine.— Carcinoma occurs in the colon more frequently than in the small intestine, and develops most often in the rectum. Carcinoma of the colon is most frequently located in the sigmoid flexure and the cæcum. In this connection the statistics of de Bovis[9] are particularly instructive.

Most carcinomata of the intestine are of the cylindrical cell glandular variety (carcinoma adenomatosum cylindro-epitheliale, Hauser[10]). Medullary carcinoma is rare; scirrhus and colloid more so. All carcinomata in this situation show a marked tendency to ulcerate and encircle the gut wall. The first explains the frequent bleeding, the second, the tendency toward stenosis, that characterize the disease. The latter is accompanied by the changes already described in connection with chronic intestinal stenosis (p. 1996). At first, the mesentery is elongated by traction, later, its infiltration causes it to shrink. The disease spreads by the lymphatics (peritoneum) and by the blood (liver); however, metastases occur late.

The *clinical picture* of intestinal carcinoma is that of stenosis of the gut, to which may be added the symptoms of tumor and those of an ulcerative intestinal process. Early in the disease one or all of the symptoms may be absent. Very often the process remains latent until suddenly, out of a clear sky, ileus appears, and section *in vivo* or *mortuo* reveals a circular stenosis, the degree of which is astonishing. In these cases the ileus is found to be the outcome of incarceration of a foreign body, or kinking of the gut has suddenly occurred. In some instances, ileus must be explained on the basis of an acute intestinal insufficiency. Occasionally the accidental discovery of a *palpable tumor* constitutes the first evidence of the disease; in this event the diagnosis is not difficult.

From this it is apparent that carcinoma of the gut is an insidious and, primarily, not a very malignant process. As long as the lumen of the intestine remains patent, the general condition of the host is not seriously impaired. In most of the cases the beginning of the disease is attended with digestive disturbances, of which constipation with occasional attacks of diarrhea is most common; these attacks become more and more frequent, and, as the lesion progresses, distention accompanies the periods of constipation; this is a suspicious circumstance. In addition to this the periods of constipation are attended with colicky pains and, not infrequently, local meteorism may be

observed. Later, vomiting occurs during the attacks; this is mostly reflex, the vomitus consisting of the contents of the stomach, but, still later, the vomiting becomes that of ileus. One may say, that the picure is one of chronic intestinal stenosis appearing in an apparently healthy person.

When the picture is fully developed, the presence of a tumor may be regarded as diagnostic; however, carcinomata in the flexures are usually not recognizable in this way until late in the disease. At about this time the *stool* becomes characteristic; it contains blood, mucus, pus, and tissue fragments, and emits a foul odor. Carcinoma of the gut does not produce pain of itself; this is caused by stenosis and is of a colicky character.

The special symptoms indicative of the location of the tumor are in every regard similar to those discussed in connection with chronic intestinal stenosis (p. 1996). The *chief three symptoms* of intestinal carcinoma are *tumor,* evidences of *ulceration,* and *obstruction.* In the absence of these the diagnosis rests on the constitutional symptoms — cachexia.

In the *differential diagnosis* the question as to the character of the local condition comes up. In this connection other tumors and the chronic inflammatory processes must be taken into account. In all instances an examination should be made before and after thorough evacuation of the bowel; in many cases a conclusion will not be reached until the tumor is exposed by celiotomy.

The *treatment* of carcinoma of the gut is that of all malignant processes, i. e., extirpation. Operative efforts at relief are contraindicated (1) when the general condition of the patient is such that the operation is likely to be followed by a fatal outcome; (2) by the impossibility of overcoming the technical difficulties, i. e., the presence of metastases and the extent and location of the disease. However, it must be remembered that when radical removal of the disease is not possible, intestinal exclusion still offers the opportunity to afford relief of the symptoms. The *radical operation* consists in resection of the tumor together with the mesentery and the regional lymph nodes; it is best performed by the three stage operation of Mikulicz (p. 1954). If there is much dilatation of the oral gut, preliminary enterostomy is permissible.

The palliative operative measures are (1) colostomy, (2) enteroanastomosis, (3) intestinal exclusion.

Mesenteric Embolism and Thrombosis.— Of the pathological processes

occurring in the mesentery, other than those already discussed, those arising in connection with embolism and thrombosis possess surgical interest; they bear a close resemblance to primary lesions of the gut and are often responsible for grave errors in diagnosis.

While the ample blood supply makes it possible for embolism and thrombosis of its arterial system to occur and not to cause disturbances, this is only true provided the main tributaries are not occluded. When the superior mesenteric artery is obstructed, there remains only the gastroduodenal and the inferior mesenteric; if the inferior mesenteric artery is occluded, blood gains access to the intestines only by way of the superior mesenteric and the median and inferior hemorrhoidal vessels. The circulation of the gut is especially damaged when the superior mesenteric artery no longer functions; this is followed by the development of a hemorrhagic infaret, involving the greater part of the small intestine. If the embolus is arrested in a branch of the superior mesenteric artery, an infaret develops in the presence of arterial selerosis. The failure of the establishment of a sufficient collateral circulation results in gangrene of the gut. Occlusion of small branches of the mesenteric arteries may be followed by perforation.

Embolism is caused by septic processes, endocarditis, cardiac valvular disease, aortitis, etc. *Thrombosis* occurs in connection with local arteriosclerosis, lues, and trauma, i. e., rupture of the artery, hematoma, etc. Of 125 cases of occlusion of the mesenteric vessels, 88 involved the arteries; of these, 79 obstructed the upper, and 9 the lower, branch (Neutra[11]).

Embolus of the mesenteric arteries is attended with severe colicky pain, soon followed by more or less blood stained diarrhea. A not inconsiderable number of the cases die in twenty-four hours; in others, the picture of paralytic ileus gradually develops. In the latter class of cases the diarrhea ceases, the abdomen becomes distended, and vomiting, which is often bloody, begins. When a restricted sector of the gut is affected, perforation is ultimately responsible for peritonitis. In rare instances a picture of moderate degree is followed by recovery; in these instances the diagnosis is doubtful.

Obstruction of the mesenteric *veins* is attended with a similar anatomical and clinical picture. Considering the influence that incarceration, infection, and portal stasis exercise upon the veins of the mesentery, it is astonishing how infrequently the condition develops to a sufficient extent to be recognized. The most frequent causative

factors are portal stasis and infection of the gut. Thrombosis is still more fatal than is embolism (Roussel[12]).

While the *prognosis* is very unfavorable, the fact that one cannot be sure of the extent of the involvement justifies the employment of operative measures of relief (Kausch[13]).

The *operative treatment* consists in resection of the damaged gut. Gobiet[14] reports 68 cases, of which 12 recovered; 2 of these were thromboses. Kausch[13] quotes a case of thrombosis involving the colon which recovered. A similar case operated upon in the clinic of the writer recovered; the lesion was located at the hepatic flexure. Robson[15] reports a case of recovery following ligature of the superior mesenteric vein.

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CHAPTER X

ILEUS

Intestinal obstruction may be divided into *mechanical* and *dynamic*. The *mechanical* is subdivided, in accord with its cause, into (1) *obturation ileus* (the lumen of the gut is obstructed without interference with the mesenteric circulation); (2) *strangulation* (besides the obstruction, interference with the circulation is the dominant factor); (3) *strangulo-obturation* (a pure obstruction ileus is followed by filling and distention of the gut, which in turn damages the mesenteric circulation, and ultimately causes gangrene). *Dynamic ileus* includes that caused by *paralysis* of the gut (following inflammatory processes and reflex nervous influences) and by *spastic contraction*.

General Considerations Regarding the Development and Symptoms of Obturation Ileus.— In order to understand the complicated manifestation incident to obturation, it is necessary to possess a knowledge of the typical processes occurring in connection with obstruction of the small and the large gut. For the purpose it is permissible to analyze these events as applied to a sudden occlusion of the lower portion of the ileum (where obstruction most often occurs) by a gallstone, a foreign body, kinking due to an adhesion, or other causes.

The moment the occlusion occurs, the gut, in an effort to overcome the obstruction, undergoes a violent muscular contraction; this is attended with severe pain, more or less shock, and expulsion of the stomach contents (*initial vomiting*). The last is due to reflex nervous influences. After this, the gut, above the hindrance, is gradually filled with fluid and gases, becomes dilated, and *peristaltic activity is increased*. At this time auscultation reveals more or less persistent intestinal noises provoked by the motion of the gut in its unsuccessful efforts at propulsion of the contents. The auscultatory sounds are produced by the motion of the gases through the fluid, the former of course rising to the highest point; when the dilatation reaches a certain degree, the sounds are changed in character. At this time, *metallic tinkling* may be elicited. Wilms[1] ascribes the presence of

metallic tinkling to tension in the gut and regards it as an important early symptom of ileus. When the accumulation of gases and fluids increases still more, so that two or three loops of gut are filled, the well known symptom of *periodical attacks of rumbling* develops. The sounds resemble the "gurgling" attendant upon pouring fluid from a bottle. The mechanism of the production of the sounds is as follows: A sector of the gut becomes completely filled; its musculature contracts and forces the contents against the obstruction; the musculature suddenly relaxes and the fluid regurgitates into the upper empty intestinal loop. These attacks occur every few minutes; *pain* begins at the moment of greatest contraction and ceases suddenly when the gut relaxes. In a thin abdomen the phenomenon may be visualized; the distended loops appear in the form of *sausage shaped protrusions* in a flat abdomen, although this may at times still be observed in a widely distended meteoric abdomen.

Intense attacks of pain, often attended with vomiting, persist only as long as the contractility of the gut is retained. Wilms[1] attributes the pain to traction on the mesentery.

To the symptoms stated, i. e., *initial vomiting, shock, pain, metallic tinkling, intestinal rumbling,* and visible *tumor,* is added, sooner or later, *fecal vomiting,* as the oral gut is increasingly filled. When the gut is filled up to the duodenum, each contraction of the musculature forces a certain amount of the intestinal contents into the stomach so that the vomitus is stained with bile and emits a feculent odor.

At the end of one or two days — following the onset of the obturation — fecal vomiting is clearly established. No case of intestinal obstruction should be permitted to reach this stage. However, at this time, so-called decomposition of the intestinal contents begins to affect the general condition of the patient. The absorbed toxic substances damage the liver and paralyze the blood vessels; this is attended with *sweating, pallor, cold extremities,* and the general picture of profound exhaustion. The eyes are shrunken and surrounded by dark rings; the outline of the nose is sharpened, its tip is cold; the forehead is covered with sweat; the pulse is rapid and feeble. During the entire disease the *lower bowel* is inactive, except that at first a small quantity of feces may be expelled. The *urine* is scanty, and the *thirst* is unbearable.

The lapse of time between occlusion of the gut and the appearance of the symptom complex of ileus may be considerable. In some cases, for instance, when the obturation is caused by gallstones, feculent

vomiting may appear in twelve to twenty-four hours; in others, this may not occur for three or four days.

The symptoms as stated must be clear in the mind of the surgeon if a fatal outcome is to be avoided. When obturation of the gut is not promptly relieved by surgical measures, the retained intestinal secretions are quickly responsible for an *autointoxication,* the lethal effect of which determines the outcome. The attacks of colic, and the attendant rumbling sounds, become less and less frequent; meteorism embarrasses respiration; and the dehydration of the body, consequent to the collection of fluid in the gut and its expulsion by vomiting, results in cardiac failure. As a rule, the *sensorium* remains clear until the end. In the last few hours of life, complete paralysis of the gut may be accompanied by cessation of the symptoms and the patient's discomfort is not great. In other instances, the picture terminates with peritonitis, the outcome of migration of the intestinal bacterial flora through the wall of the gut, or, which is more likely, with perforation (necrosis from circulatory disturbances; the *distention ulcer* of Kocher).

So far, obturation of the ileum has been discussed. The course followed in these cases does not vary perceptibly when the lesion is located in the cecum or jejunum. When, however, obturation occurs just below the *duodenojejunal flexure,* i. e., in the upper jejunum, but little of the gut is distended and meteorism is not marked. Persistent vomiting occurs early and as the quantity of arrested contents is not large, the patient's general condition is not rapidly impaired. However, the vomitus is likely to be feculent in character. The presence of bile and pancreatic juice differentiates the condition from pyloric stenosis.

Obturation of the small gut is caused by gallstones, enteroliths, worms, and foreign bodies; also by tumors outside the gut (pressure), especially those located in the true pelvis; abscesses and cysts, and tumors developing in the *intestinal* wall, such as lipomata, adenomata, myomata, and sarcomata (often by invagination). However, none of these is as frequently responsible for acute ileus as are *adhesions and bands* such as develop sequentially to appendicitis and salpingitis, especially after operations. While the various causes are more extensively discussed farther on, it is permissible to accentuate here the fact that all of the causative factors mentioned provoke a similar clinical picture and that only a very accurate history affords an opportunity to make diagnostic distinctions in this regard. This justifies

the conclusion that in most instances the surgeon must be content with establishing the diagnosis of ileus of the small intestine, and leave its exact cause to be cleared up during the operation.

Obturation of the colon possesses distinguishing features. In occlusion of the gut near the cecum the cause of the obturation is usually palpable. The symptom complex is identical with that of obturation of the lower ileum. It differs distinctly, however, when the occlusion is in the *more advanced portions of the colon.* In these instances, the picture varies, first, in accord with whether the *oral or aboral portion of the colon* is obstructed; and second, whether the *ileocecal valve* is operative in a retrograde sense or not. In obstruction at the hepatic flexure and transverse colon the small intestine fills up when the valve is *insufficient;* the right abdomen is distended, the cecum is palpable, and typical attacks of colic and gurgling are present in the same way as though the cecum and the small gut were obstructed. When the valve is competent, the contents is arrested in the cecum, and as this organ is capable of great distention, a condition of affairs called *isolated distention of the cecum* is developed. This often leads to perforation, so that in two or three days fatal peritonitis occurs, or the cecum is so much damaged that its removal is necessary.

Obstruction at the *splenic flexure,* the *descending colon, and the rectum,* is usually due to kinking or to carcinoma, so that the *wall of the gut* is always much *thickened.* When acute obturation occurs in these situations, in the absence of thickening of the wall of the gut, the oral colon may usually be outlined through the abdominal wall in the form of a bow curving above the level of the navel. Percussion elicits metallic tympany in the line of the transverse colon and cecum; but there is no visible increase in peristalsis. When, however, the gut is hypertrophied (in carcinoma and stricture), complete obturation is attended with *increased peristalsis,* plainly discernible through a thin abdominal wall; the peristaltic wave moves from right to left. In these cases the cecum is also markedly dilated and, even in corpulent persons, is palpable.

The distinguishing feature of *obturation of the lower portions of the large gut* is the *gradual development of the disturbances;* initial vomiting may be absent; abdominal distention is very gradual, so that marked meteorism does not appear until the fifth or sixth day; peristaltic sounds are likely to be absent. When the colon is widely dilated percussion elicits metallic tympany. The colon is rarely obstructed by gallstones or foreign bodies; in most cases the obturation is caused

by *kinking* or by the *sudden occlusion* of an already existing stenosis (usually carcinoma). *Kinking* occurs most frequently at the splenic flexure. Carcinoma is most often located at the junction of the descending colon and the sigmoid flexure.

General Considerations Regarding Strangulation of the Intestine.—The *symptom picture of strangulation* varies with the *intensity of the ensnaring force, reaction* on the part of the abdominal nervous system, and with the *extent* of the strangulated gut. Taking the incarceration of a loop of small gut, in a defect in the omentum, as a type, the onset is attended with expulsion of the stomach contents and *severe pain.* The initial vomiting is followed by an interval of from two to four hours, when it begins again and is from then on persistent; at this time the vomitus contains bile, but is not feculent. The abdomen is not distended, as a rule, until the second day; on the contrary, it is likely to be flat, and, if examined, the primarily ensnared intestinal loop is likely to be palpable in the form of a *circumscribed tumor* presenting *localized meteorism* (v. Wahls symptom). In most cases the mass is located in the region of the cecum, where strangulation most frequently occurs. Strangulation may be attended with *reflex tension of the abdominal muscles;* this, as a rule, is diffuse and ascribable to an effort to relieve pain.

As compression of the nerves of the strangulated loop provokes reflex paralysis of the entire gut, rumbling and gurgling sounds are not produced, so that auscultation is valuable only in a negative sense at this stage of the lesion. The tension of the abdominal wall may convey the idea of *peritonitis;* however, the absence of fever should obviate error in this connection.

When a large sector of the gut (30 to 40 cm.) is strangulated the coincident infarction is attended with the formation of a bloody exudate in the abdominal cavity; in from twelve to twenty-four hours, this may be recognized by physical examination. On the second or third day the transudate causes paralysis of the gut, and local resistance and meteorism is obliterated. Strangulation of an isolated sector of the gut may be diagnosticated during the first, or perhaps the second day, when *persistent vomiting and pain, local resistance, and tension of the lower abdomen is not attended with fever and the entire gut is inactive.*

When peritonitis develops (usually the third day) at the site of the ensnaring, all the symptoms of strangulation are effaced and an exact

diagnosis is impossible. In these cases the surgeon does not gain anything by delay; the picture will not become clearer as time goes on.

Strangulation of *large portions of the gut* is expressed by a very different clinical picture. In this event, a large portion of the blood is collected in the excluded intestinal loops and the picture resembles that of internal bleeding. In addition to this, massive strangulation is attended with copious bloody effusion into the peritoneal cavity, and this, in a few hours, produces complete intestinal paralysis. In this class of strangulation the arterial blood supply is maintained, while venous return is obstructed, which accounts for the rapid anemia. The picture of loss of blood is especially defined in cases of congenital disturbances of the small gut, in which this in its entirety is twisted at the origin of the mesentery. In these instances the fatal outcome is only a matter of a very short time.

Having established the difference between strangulation of small and large sectors of the gut, there remains a third class which *begins with obturation,* although the cause of the obstruction consists in a strangulation. In order to understand this the surgeon must realize that while the general symptoms of strangulation occur in most cases in which the gut is suddenly snared (hernia for instance), in others the picture is attended with that of gradual obstipation, vomiting, increased peristalsis, etc. In the one case, the nervous system of the abdomen reacts most intensely and the entire intestinal tract is paralyzed; in the other, the disturbances are purely local and may escape recognition (especially in femoral hernia in women). This is most likely to occur in old persons and is equally true with regard to *internal strangulation.* That is to say, internal strangulation may present the symptoms of *obturation* and argue for early operative interference as soon as a tentative diagnosis is established. In this connection, we have in mind especially *volvulus* of the sigmoid, small gut, and cecum.

The *several forms of volvulus* present a varying clinical picture, in accord with the extent of the twisted gut and the degree of the torsion. Complete obturation is usually preceded by attacks of incomplete obstruction — a valuable factor in the diagnosis. The subject is more fully discussed farther on.

Strangulo-obturation presents an atypical clinical picture. It is characterized, at first, by the symptoms of obturation, i. e., increased peristalsis, gurgling and rumbling sound, metallic tympany, meteorism, and obstipation; but on the third or fourth day the picture changes to

that of strangulation with intestinal paralysis and bloody ascites. This twofold picture occurs in connection with *long bands,* such as a fixed appendix or tube, Meckel's diverticulum or stretched adhesions which form a large ring. When a certain degree of tension occurs in these bands, only the lumen of the gut is obstructed and the symptoms of obturation develop. At first, the mesentery is not compressed and the circulation of the gut is not impaired. As the gut is gradually distended, it drags heavily upon the mesentery which is then strangulated. Thus is produced paralysis of the gut with its attendant symptoms of strangulation, i. e., meteorism, ascites, fecal vomiting, etc., followed by a fatal outcome in two or three days, unless relief is afforded.

The manifest *toxicity of the contents* of the oral sector of the obstructed gut has led to experimental study intended to elucidate the character and the source of the toxin. In this connection, the work of Whipple,[2] Lynch and Draper,[3] and Sweet[4] is of value. Eisberg and Draper,[5] basing their conclusions on the work of others and their own not inconsiderable experimental data, state "Whatever the composition of the toxin of the intestinal obstruction, which we believe to be of the same nature as the known endocrin secretion, partly because of the similarity of the symptoms after parathyroidectomy or duodenal obstruction, all observers agree that its most active manifestations occur in connection with duodenal occlusion. There is a point in the second portion of the duodenum at which acute obstruction causes death more rapidly than elsewhere in the gut. Oral or aboral to this line there is a proportionate decrease of obstructive toxicity. Lynch has suggested that there is normally some biochemical product brought down from above similar in nature to prosecretin. With this view we are in hearty accord.

"Duodenal transplantation definitely shows that the duodenum and its appendages, singly or collectively, produce the lethal agent which causes death in intestinal obstruction.

"A proteose may be isolated under certain conditions, which, when injected, causes death."

These conclusions argue for drainage of the gut high up, as near as is feasible to the duodenum in all cases of ileus. They are borne out by the clinical observation that the establishment of a fecal fistula which drains the oral sector of obstructed gut gives the best operative results.

The establishment of three general forms of mechanical ileus —

obturation, strangulation, and strangulo-obturation—opens the way to a consideration of the special forms of obturation ileus.

Occlusion of the Gut by Adhesions.— Adhesions develop between the various sectors of the gut, and between the gut, omentum, abdominal wall, and other organs, when the peritoneum is damaged. The causes of adhesions are first (and foremost), inflammatory processes (appendicitis, perforations of stomach and gut, cholecystitis, salpingitis, etc.); and second, mechanical damage during operations. The latter occurs most frequently sequential to so-called gynecological operations. Chemical trauma is due to the action of antiseptics and the thermocautery. Finally, ligature of especially large stumps of tumors or of the omentum is often responsible for the formation of adhesions.

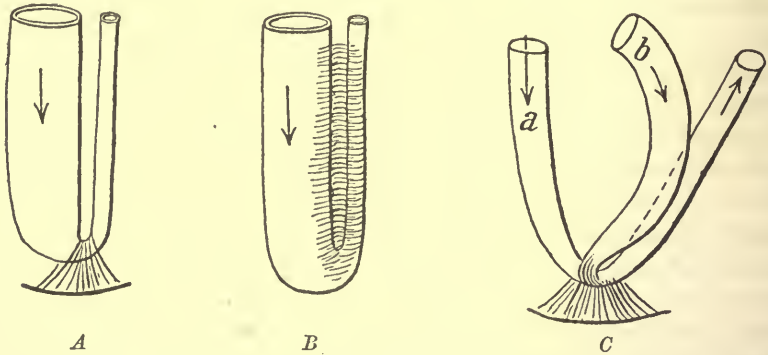

FIG. 909.— MECHANISM OF INTESTINAL OBSTRUCTION BY ADHESIONS.

A, Gut adherent to abdominal wall. *B*, Adhesions between two loops of gut. *C*, Twisting of oral gut; loop adherent to abdominal wall (wring obstruction) (Wilms).

The deligated tissues act like a foreign body in the same way as *tamponade and drainage*. By far the largest number of cases of obstruction occur in connection with the organs located in the lower sectors of the abdomen.

The influence of adhesions varies with the character and extent of fixation of the small gut. Despite the multiplicity of the lesions which make schematization in this connection difficult, the following presentation of typical forms would seem to be permissible: (1) Adherence of the side of the gut wall, causing a valvular occlusion; (2) when adhesions between two loops of gut cause obstruction; (3) when twisting of the afferent limb causes obstruction (wring obstruction — previously called volvulus of the gut on its axis). The three forms are

shown in Fig. 909. It is of course necessary that the oral limb of the fixed loop be distended with gas or fluid; thus the gut at the point of fixation is kinked, or the distention of the afferent drags the efferent toward itself and flattens it out (Fig. 909B). This relatively simple form of obstruction is less frequent than that shown in Fig. 909C, in which the distended oral limb of the fixed loop changes its position and approaches the point of the adhesion. In this way the distended loop is not only kinked at the point stated, but is also rotated on its axis, and thus torsion completes the occlusion. This form is called *wring occlusion* because the motion of the oral limb is similar to that employed in wringing out clothes (Wilms[1]).

Intestinal occlusion of the forms just stated may be spontaneously corrected. This is shown by the fact that many cases of intestinal obstruction give a history of a greater or less number of previous attacks, and celiotomy reveals just such a condition of affairs as is here described. When spontaneous correction is to be awaited, it is impossible to say; however, this is certain, it can be expected only when peristalsis suddenly ceases; for instance, when the bowel has been immobilized by an opiate — a dangerous maneuver.

The *diagnosis* of obturation by adhesions is based on the clinical picture already presented; however, it is permissible to accentuate the importance of obtaining a history of previous attacks and of a previous possible causative lesion or operation.

The *treatment* is surgical in principle. Under especial circumstances, it is justifiable to make a brief attempt to overcome the obstruction by gastric and colonic lavage, the application of heat to the abdomen, and the administration of small quantities of morphia. The last may be used only when the surgeon is certain that the symptoms are caused by adhesions. This is so remote that perhaps one is justified in stating that the administration of an opiate is only justified for the purpose of relieving pain when the decision to operate has been arrived at.

The *surgical treatment* varies with the time of the occlusion and the age and general condition of the patient. In *late cases*, when the diagnosis of obturation of the small gut (probably by adhesions) has been made, it is best to establish a *fecal fistula* in either the right or the left lower abdomen, and to attack the site of the obstruction later. As obstruction of this sort occurs most often in the region of the cecum, most surgeons open the abdomen through the right rectus muscle. The corrective operation, whether primary or secondary, is

formulated in accord with the methods discussed in connection with abdominal operations (chap. iii, of this part).

Postoperative Ileus.— Postoperative ileus occurs between three and eight days after celiotomy; it is caused by fibrinous adherence and paralysis of the gut, which hinders its peristalsis.

Ileus following operations may be due to *reflex* and to *inflammatory* causes. The former has already been discussed in connection with acute dilatation of the stomach and gut (p. 1915). At this time consideration is restricted to localized ileus, the result of mechanical and thermal influences, which produce partial paralysis of one or more sectors of the gut, with or without fibrinous agglutination.

After the operation the patient recovers from shock and for two or three days does not present defined disturbances; however, there is not the satisfactory "tone" in the picture as a whole, which the experienced surgeon finds difficult to explain, but which, nevertheless, is patent. In most instances the *pulse* shows a tendency toward *periodic acceleration,* the facial expression is "flat," the mental attitude indifferent. While the writer is conscious of the indefiniteness of this description, it may be sufficiently clear to constitute a warning to those laying their first course in the shoaling waters of major surgery.

After the second or third day the pulse is persistently accelerated and loses its tone; the patient has a feeling of fullness and discomfort in the abdomen; auscultation elicits evidences of increased intestinal peristalsis. Presently, colicky pains develop and the hideous surgical "family skeleton" — fluid vomiting — stalks into the picture. The temperature is only moderately elevated; this does not mean peritonitis and possesses none of its characteristics. It is probable that the trauma to the gut, during the operation, is the most important causative factor in this form of ileus.

In postoperative ileus the intestinal paralysis is attended with kinking of the gut, produced in the same way as that occurring in connection with occlusion by adhesions, and, therefore, measures which increase peristalsis only increase the mechanical obstacle. This conception reduces the indications for *treatment* to those already described, i. e., gastric and colonic lavage; application of warmth to the abdomen; and, unless relief is soon afforded, the establishment of a *fecal fistula,* oral to the obstruction, and secondary liberation of the gut.

When one considers the mechanism of the occlusion, it is easy to see how the fistula not only gives egress to the intestinal contents, but also makes possible the spontaneous correction of the kink and reëstab-

lishment of the intestinal flow; this is shown by clinical experience. As a rule, the fistula closes spontaneously; if this does not happen, its closure is a simple matter.

Obturation ileus of the large gut is favored by its partial fixation; it, therefore, occurs most frequently at the *hepatic* and *splenic flexures* and, if the transverse colon is particularly enteroptic, this is also likely to be kinked. The presence of a so-called cecum mobile also favors kinking of the large gut when the latter is not abnormal. Displacement of the transverse colon may also be due to the drag of an adherent omentum, which produces a kinking at one or both of the flexures.

The *clinical picture* depends upon the patency of the ileocecal valve. If the valve is insufficient, the symptoms are those of obturation of the small gut; if it holds, the picture is that of isolated cecal distention.

The *treatment* consists in resection of the damaged cecum, and entero-anastomosis between ileum and transverse colon in cases in which the general condition of the patient permits; when the latter is menacing, a cecal fistula may be established and the correction made later.

Obturation at the splenic flexure is frequent. It usually occurs in elderly persons in whom a previous chronic inflammatory process has existed in the colon. These cause the oral and aboral sectors contiguous to the flexure to become adherent, which finally leads to obturation by the mechanism already described.

The *clinical picture* is that of gradual obstruction of the large intestine, and cannot of course be differentiated from that of an occult carcinoma.

The *treatment* consists in the establishment of a fistula in the transverse colon; or a colocolonic entero-anastomosis (exclusion of the splenic flexure). In a not small proportion of the cases, the high fixation of the transverse colon by means of the fistulous opening permanently corrects the kinking (Wilms[1]).

Obturation Ileus from Occlusion of the Intestinal Lumen.— Congenital intestinal occlusions are due in part to inflammatory processes and in part to twisting and snaring in fetal life. Atresias of this sort are usually multiple. As a rule, the child dies after a few days of vomiting, meteorism and colic. Surgical efforts at relief are not well borne; however, occasionally the establishment of a fecal fistula is followed by recovery.

Occlusion of the lumen of the gut by collections of feces and entero-

liths occurs most frequently in connection with previously existing *kinking* or *carcinoma.* Genuine obstruction of this sort presupposes (1) hard thick masses of feces; (2) an atrophied thin intestinal muscularis. A thick hypertrophied muscularis is not attended with the formation of scybala, even when the hindrance is located low down in the colon. Palpation of the thickened fecal masses establishes the *diagnosis.* The *treatment* consists in colonic lavage preceded by high oil enemas.

Obturation by enteroliths occurs in connection with the ingestion of hair, plant fibers, and grain shells, also after prolonged administration of chalk, magnesia, bismuth, etc. The *clinical picture* is similar to that of occlusion by gallstones, and kinking.

Obturation gallstones occurs four times as often in women as in men. As a rule, the stone lodges in the lower ileum. It gains access to the gut either through the common duct or after perforation of the adherent gallbladder into the gut. The site of fixation of the stone determines the character of the clinical picture.

The *diagnosis* is possible only when the presence of gallstones has been previously determined. Of itself, obturation by gallstones presents the ordinary picture of ileus, except that its rapid development arouses the suspicion of strangulation.

The *treatment* is based on the principles laid down in connection with obturation ileus.

Ileus due to foreign bodies and worms presents the clinical picture of obturation ileus. The *diagnosis,* unless a history of the causative factors is obtainable, is not made until the abdomen is opened for relief of the obstruction. In this event, removal of the agent, temporary fistula, resection of the gut, or entero-anastomosis is performed in accord with the indications as previously discussed.

Obturation of the gut from compression, i. e., movable organs (such as the liver, kidney or spleen) cysts and tumors, occurs in connection with the small and large gut. In some instances, ileus is primary, in others the tumor or cyst is recognized early, so that the establishment of the obstruction may be ascribed to the presence of the abnormality. Tumors arising in connection with the omentum, mesentery, gut or kidney, of sufficient size to cause ileus, are readily palpable; those located in the pelvis (rectum, bladder, and female organs of generation) are also usually discoverable by rectal or vaginal examination. The *symptoms* of compression occlusion are similar to those of obturation ileus.

Arteriomesenteric ileus (so-called duodenojejunal obstruction) is caused by compression by the mesentery of the small gut, especially the superior mesenteric artery. The pressure of the mesentery is the outcome of the drag of the gut, which is prolapsed into the pelvis, and this is enhanced by dilatation of the stomach, which forces the small gut downward. This assumption leads to the conclusion that the primary lesion consists in occlusion of the duodenum, and that *acute dilatation of the stomach* is secondary. On the other hand, there is no little evidence available

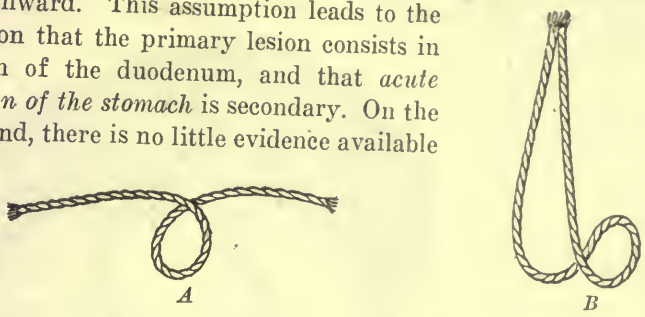

FIG. 910.—STRANGULATION BY SNARING.

A, Snare formation by a long band. *B*, Figure of eight snare (Wilms).

which would seem to show that the reverse is also true. Most surgeons lean toward the last explanation. The *clinical picture* is already taken up in connection with acute dilatation of the stomach (p. 1915).

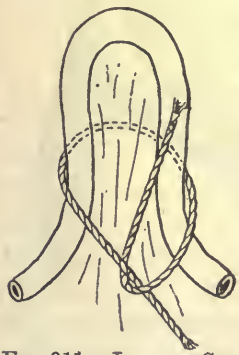

FIG. 911.—LOOP OF GUT SNARED BY A LONG BAND.

Ileus caused by changes in the wall of the gut is the outcome of cysts and tumors developing in this situation. Large tumors may obstruct the gut by pressure; but, as a rule, the occlusion is the result of invagination. The peristalis attempts to force the gut into the lumen of the gut, dragging along the wall of the latter; when the invagination thus produced reaches a certain extent, the gut is occluded. The subject is taken up more extensively in connection with intussusception (p. 2025). The tumors concerned in the cases so far reported are *lipomata, isolated cysts, polypi, adenomata, myomata,* and *sarcomata.*

Strangulation Ileus.— The general considerations with regard to strangulation have already been taken up. At this time the special forms will be discussed.

Apertures in the mesentery, the mesocolon, the omentum or those formed by adhesions are often entered by loops of gut, especially those

of the small intestine. If the aperture is small the vagrant loop of intestine is immediately subjected to elastic strangulation. In other instances a single loop enters and is not strangulated until its aboral limb is drawn farther and farther into the snare, so that the entire sector of the gut is too large for the opening. Additional gut enters through the opening, when the peristalsis of its aboral limb is hindered, and dilates at the ring; this causes the aboral limb to draw more oral gut with it.

This form of strangulation, in which usually large sectors of gut are ensnared, occurs in connection with bands and is attended with the clinical picture of strangulation (p. 2013). Narrower constricting rings also occur in connection with long bands when snares or kinks are formed. (Figs. 910–911).

In strangulations in apertures the gut often makes a turn of 180 degrees, i. e., a volvulus is formed; this increases the subsequent damage to the ensnared gut.

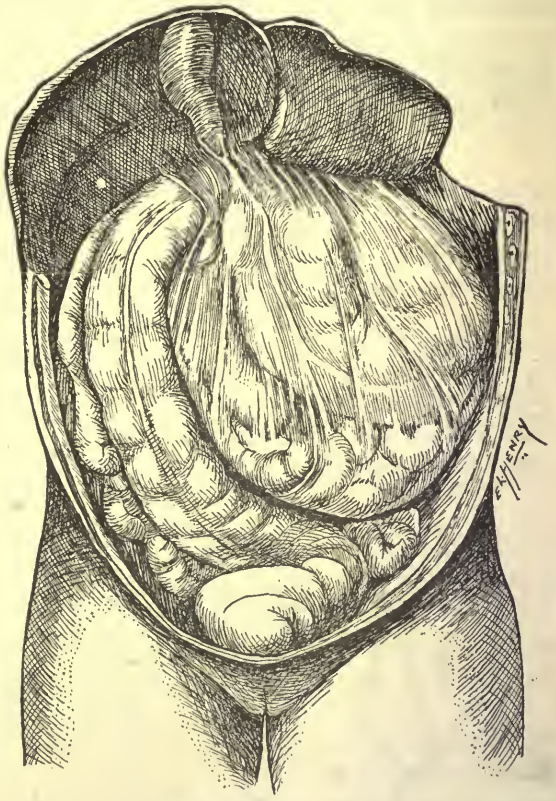

Fig. 912.— Hernia Through the Foramen of Winslow.
Lesser cavity filled by both small and large intestine.

The symptoms of this form of strangulation have already been described (p. 2013).

In strangulation of a *hernia into the foramen of Winslow* the gut enters the lesser peritoneal pouch behind the biliary passages (Fig. 912).

Owing to the large size of the ring, *hernia into the recessus duodeno-*

jejunalis usually takes on the form of obturation, rather than that of strangulation.

Occasionally the gut enters the openings located at the *origin of the mesentery*. This also happens at the *cecum*, especially into the inferior *ileocecal fossa* or the *ileo-appendicular*, the *cecal* and the *subcecal fossae*. The prevesicular space, the pouch of Douglas, and the intersigmoid fossae are rarely entered by the gut. Operative exposure of a hernia of this sort invites closure of the opening by suture.

Ileus in connection with Meckel's diverticulum occurs as an *invagination* of the gut. In other instances, the diverticulum is *rotated* and causes an obturation, or the loop to which the diverticulum is attached is rotated (volvulus of the small intestine). The tip of the diverticulum may become adherent and form a *ring*, within which the small gut becomes strangulated. Finally, a long slender diverticulum may snare a loop of gut, as happens in connection with long bands.

Strangulo-obturation occurs when the greater part of the small gut is propelled over a tense residual diverticular band. As more and more of the gut crosses the band, the latter takes on the form of a ring, which slowly obstructs the obtruding intestine, and the *clinical picture of obturation* is gradually developed. As the thus engaged gut fills, the mesenteric circulation is interfered with, and on the third or fourth day the picture of *strangulation* is superimposed upon that of obturation, i. e., the total picture is that of strangulo-obturation.

The *treatment* is similar to that of strangulo-obturation described in the general considerations.

Volvulus.— Twisting of the gut (cecum and sigmoid) is most often observed in Russia and Sweden; this argues for a congenital disposition in this connection. Rotation of the small intestine is caused, in part, by a congenital elongation and narrowing of the mesentery. In these cases the prompt occlusion of the circulation results in a rapid fatal outcome. The lesion presents the *clinical picture* of strangulation of a large sector of gut.

The small intestine usually rotates with the clock; the cecum and sigmoid may be twisted in either direction.

Volvulus of the cecum occurs as the result of a *faulty fixation* of itself and the contiguous ascending colon, precipitated by distention of the part by gas and fluid.

The *diagnosis* takes into account previous attacks of colic and vomiting, associated with swelling of the right lower portion of the abdomen.

Volvulus of the cecum does not, as a rule, present the grave picture of strangulation, but appears in the form of *obturation*.

The *course* of the lesion is slow, so that surgical efforts at relief are likely to be successful as late as the third or the fourth day. When the cecum is not much damaged, its position may be corrected and the organ fixed to the posterior abdominal wall; in more severe cases the cecum must be resected. When the oral gut is distended with toxic contents, the establishment of a *fecal fistula* is indicated.

Volvulus of the sigmoid flexure is more common than that of other portions of the gut; it is determined by *shrinkage of the mesentery* in the presence of a *congenitally enlarged flexure* (Wilms[1]). The deformation of the mesentery may be due to chronic inflammatory processes in the sense of lymphatic irritation coincident to prolonged obstipation.

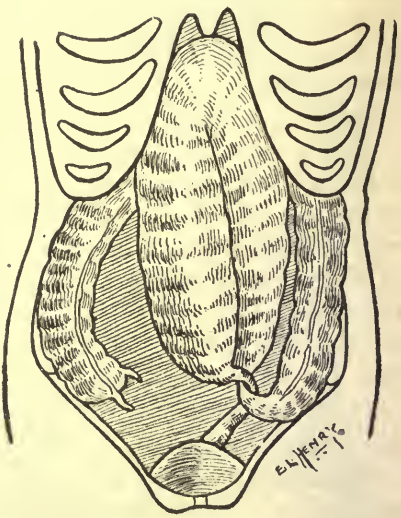

FIG. 913.— VOLVULUS OF SIGMOID: TWIST TOWARD THE LEFT.

The acute torsion is usually preceded by more or less severe attacks of colic provoked by partial twisting, which is spontaneously corrected; this indicates that the final rotation is the outcome of repeated damage to the mesentery. When complete torsion occurs, a tumor, sometimes of enormous dimensions, is developed. The abdomen becomes greatly distended and even the arch of the ribs may be increased in its arc. This tumorous distention emits a high pitched tympanic percussion note; peristaltic sounds are absent. *Pain* is referred to the sacral region, while in other forms of volvulus pain is more marked in the region of the navel. The general condition of the patient, contrasted with the degree of tympany, is good. Occasionally, however, the onset of the torsion is attended with the picture of strangulation. Interference with the mesenteric circulation is of course of great prognostic import. In this event, the picture is that of strangulation with its attendant shock, vomiting, etc.

Treatment with enemas and catharsis occasionally succeeds in correcting the twist; this is rare and these measures must not be long per-

sisted in. The surgical treatment consists in left rectus celiotomy with correction of the rotation; mobilization of the sigmoid by longitudinal incision of the mesentery (which is of doubtful utility); or, which is desirable, the sigmoid is resected. In the event of delay in opening the abdomen, the establishment of a *temporary fecal fistula* may be necessary.

Knotting of the gut occurs only between a large flexure and the small gut. This does not mean that a flexure embraces the small gut as a "clinging vine entwines the towering pine." The small intestine slips behind the root of a flexure and by peristalsis more and more of the former is forced around the base of the latter. In this way the gut is obstructed, becomes distended, and the two loops grasping the root of the flexure are strangulated.

FIG. 914.—ILEOCECAL INVAGINATION.

The lesion presents the *clinical picture* of rapid internal strangulation and is fatal in a short time. It cannot be diagnosticated except by early celiotomy.

Intussusception. — Intussusception (intestinal invagination) consists in the telescoping of one part of the gut into the part above or below it. The former is rare. The portion of gut receiving the invaginated sector is called the *intussuscipiens;* the telescoped part, the *intussusceptum.* The apex is that part of the intussusceptum projecting farthest into the gut, the neck is the constricted portion where the intussusceptum passes into the intussuscipiens. Thus the invagination consists of three intestinal tubes; the inner two have their serous surfaces apposed and the third (outer) receives and ensnares (at the neck) the other two. A double invagination in which an intussusception becomes a part of an additional invagination occurs occasionally; in this event the lesion consists of five cylinders.

The menace in the condition, other than the mechanical obturation, consists in interference with the mesenteric circulation, which leads to strangulation. This occurs more frequently in the *acute form* of the

lesion (in children); in the chronic form adhesions between the apposed peritoneal surfaces may produce a permanent invagination, or the intussusceptum may slough off and the continuity of the gut be spontaneously restored. This is rare in children, but has been observed in the adult (Hermes[6]). Kanavel[7] reports a remarkable case in this connection.

In the *etiology,* three factors are important, (1) the difference in diameter of the small and large intestines; (2) prolapse of the mucosa of the ileum; (3) abnormal motility of the mesentery. Wilms[1] considers insular spasm of the gut, due to chronic irritation, the most important causative factor. Tumors have been responsible for invagination in a number of instances; in this connection the statistics of Weiss[8] are instructive. The association of chronic inflammatory diseases of the gut (ulcer, dysentery, tuberculosis, etc.) is unquestioned (Clubbe[9]).

The *diagnosis,* in the acute form, is made easy by the occurrence of *blood in the stool,* together with the presence of a palpable tumor. In other regards, recognition of the lesion is not possible until exploratory celiotomy, made for the relief of the obstruction, uncovers the character of the lesion.

Chronic invagination, which usually involves the large gut, presents the clinical picture of chronic ileus, plus that of dysentery, together with a palpable tumor in the course of the colon.

The *surgical treatment of intussusception* takes into account disinvagination, which is possible only early in acute cases, and *resection of the gut.* The establishment of a *fecal fistula* may be necessary when the oral gut is seriously impaired.

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CHAPTER XVI

APPENDICITIS — PERITYPHLITIS

The term *appendicitis* has established its right by usage. *Perityphlitis* is more indicative of the processes having their origin in the appendix, but does not convey the idea of the important influence the latter exercises in this connection; therefore, while the writer is conscious of its inadequacy, the term appendicitis is here retained.

Having in mind the difficulties entailed in an effort to *classify* the many inflammatory processes originating in connection with the appendix, it would seem wisest to base a classification on a macroscopic pathologico-anatomical basis; this is more purposeful than an attempt to classify the subject from an etiological or clinical standpoint. The involvement of the peritoneum gives the inflammation its characteristic impress and determines the clinical symptoms. This is true whether only the peritoneum covering the appendix, that immediately contiguous to it, or this membrane in its entirety is inflamed. Extension of the inflammation to the peritoneum is constant, except in very slight transient catarrhal conditions of the mucosa (*appendicitis simplex*).

Two main groups may be differentiated: *acute* and *chronic appendicitis*. It is probable that acute appendicitis is almost always preceded by insidious inflammatory changes in the appendix, which create a condition of affairs favoring the acute attack. An acute attack — *la crise aigue* of the French — is, therefore, only a stage in the entire disease. It may recede without disappearance of the chronic condition; this accounts for the recurrences so frequently observed, but does not mean that appendicitis does not occur in perfectly healthy persons. The chronic inflammatory condition may not provoke symptoms.

The *etiology* of appendicitis has been much cleared up in the last ten years.

The *congenital or inherited anatomical relationship between the cecum and the appendix* constitutes a disposition to the development of

inflammations. It also explains the preponderance of inflammatory processes developing in the cecum and appendix as compared to other diverticula or sectors of the gut. The position of the cecum, and, consequently, the appendix, varies widely. The range of intestinal excursions, especially of the cecum, depends upon the degree of embryonic coalescence between the mesenteric lamellae and the contiguous portions of the peritoneum. If this coalescence is faulty, the cecum and the ileum possess a mesentery in common — a so-called mesenterium ileocecale, or ileocolic commune. A cecum distended with gas reaches to the linea alba at the symphysis; one filled with semisolid contents sags into the true pelvis; an empty contracted cecum lies on a level with the crest of the ileum and is covered by loops of ileum. These varying positions have been verified by opening the abdomen and by the Röntgenogram. It has been observed that the position of the cecum varies in accord with the quantity and character of its contents, the position of the body, and, above all, in accord with the gases formed in the colon. The cecum is concerned in thickening the chyme; it is the site where bacterial flora is at its maximum, and is actively engaged in nutritional absorption. It is also important that in a certain number of instances the mesentery of the ascending colon is atypical and arises from an abnormal portion of the posterior abdominal wall. In this event, the cecum and a part of the ascending colon floats freely about in the region of the navel, or it may migrate to the left of the navel. This is usually attended with other abnormalities, and types of the intestinal tract. It is not unusual for the cecum and its appendix to be tilted toward the diaphragm covering a corresponding part of the ascending colon. The appendix is often elongated, its tip reaching sideways and upwards to the liver.

All of this indicates that the much heralded *cecum mobile* is not pathological in itself; its frequency argues strongly against it. Nor may kinking and habitual torsion of the cecum be regarded as more than a predisposing causative factor. Not until inflammation (*typhlocolitis*) develops do clinical symptoms become manifest. It cannot be denied that a movable cecum functionates better than a fixed one.

The point of origin of the appendix is at the midline of the cecum, at the termination of the anterior longitudinal band, which is the surgical guide in this connection. It varies from six to twenty-eight centimeters in length and is usually curved in outline.

The so-called *"family disposition"* is covered by the anatomical

arrangement spoken of. The similarity in this regard has been observed by surgeons operating upon several members of a family.

As a rule, the appendix is located intraperitoneally; occasionally, a retrocecal appendix is covered only in part by peritoneum. *Three main positions* of the appendix are differentiated, the *pelvic,* the *ileocecal,* and the *lateral cecal. McBurney's*[1] *point* is located at the middle of a line drawn from the right anterior spine of the ileum to the umbilicus. It corresponds to the most frequent location of the ileocecal valve and has great practical importance (Murphy[2]).

So far the predisposing factors concerned in appendicitis have been considered; however, at this writing, appendicitis is universally regarded as an infectious process caused by bacteria. The bacteria are either pathological when they enter the intestinal canal or may exist normally in this situation and become pathological as the outcome of certain conditions. The excitant may reach the appendix through the blood (hematogenous infection). Empyema of the appendix occurs, occasionally, in connection with pneumonitis. The hematogenous origin of appendicitis is indicated by the isolation of bacteria not usually found in the gut. An appendicular cyst is especially susceptible to hematogenous bacterial invasion. It is probable that this form of appendicitis is most common in children who are especially susceptible to acute infectious diseases (the exanthemata, tonsillitis, etc.). However, hematogenous infection plays a minor rôle in the etiology of appendicitis; as a rule, the disease is *enterogenous* in origin. The appendix takes part in all chronic and acute infections of the intestinal canal, especially in connection with typhlocolitis. In the latter instance appendicitis is a secondary disease; the inflammatory process recedes in the cecum and colon; but, as the appendix is not drained, the infection is localized in its tissues. While this localization occurs most often in the appendix, a similar condition of affairs also develops in other diverticula, especially in Meckel's diverticulum and in the appendices epiploicae of the sigmoid flexure, and is attended with a clinical picture closely resembling appendicitis. When the appendix has repeatedly taken part in an inflammation of the colon, its anatomical peculiarities are responsible for the maintenance of a condition of chronic irritation which, upon slight provocation, gives rise to an acute inflammatory process.

While the *bacterium coli* is not responsible for the greatest number of inflammations of the appendix, its flora soon exceeds that of the others present. Streptococcus infections are always severe, and are

especially menacing as regards outcome. In children, pneumococci are likely to provoke fatal infections. In these cases the appendicular involvement is often a part of a general pneumococcus enteritis; however, removal of the appendix is frequently followed by recovery. In addition to the two bacteria mentioned, a number of forms of bacteria are found, namely, diplo-, staphylo-, and other cocci. Anerobic bacteria play an important part in appendicitis. It is certain that no one bacterium is responsible for the disease. Distinctive clinical pictures do not develop in accord with the special bacteria concerned.

Among the many etiological factors adduced in connection with appendicitis, the acute infectious processes have been included. Of these, influenza is most often regarded as responsible for the disease (Lucas Championnière[3]), a viewpoint which would seem to be supported by clinical observation. Ochsner[4] considers that indiscretions in diet may precipitate an acute attack; this is not proved.

As *fecal concretions* are also found in normal appendices, most authorities consider that they are simply responsible for some of the destructive changes seen in a severe acute attack. Sonnenburg[5] thinks that irritation from a fecal concretion may precipitate an acute attack. *Foreign bodies* (pins, needles, nails, shot, fish bones, etc.) are occasionally found in the appendix, and may, of course, be responsible for the disease.

The influence of *obliteration* of the appendix is not settled. According to late investigations, two forms of obliteration occur, the inflammatory, which predominates in early life, and the non-inflammatory of later life; in a part of the latter the chronic inflammatory processes must be taken into account.

With regard to *traumatic appendicitis*, it would seem to have been shown that a normal appendix cannot be inflamed by injury, even when concretions are present. In no case may a traumatic cause be ascribed to the process, if it begins more than twelve hours after receipt of the injury; however, Murphy[2] and others do not subscribe to this view. Sonnenburg[5] says that while true traumatic appendicitis does not exist, there are cases of appendicitis that are made worse by trauma.

The Pathological Anatomy of Appendicitis — ACUTE APPENDICITIS.— Primarily the mucosa of the appendix is attacked. *Microscopically*, the mucosa is grayish yellow in color, swollen, injected, presents small hemorrhagic areas, and is covered with secretion. The other layers of the wall are swollen, the vessels of the serosa are engorged. There is

not a unaminity of opinion with regard to the microscopical changes. Sonnenburg,[5] after analyzing the views of others, states that a definite law applicable to all cases cannot be established. The analogy of the disease to similar processes in other portions of the gut would indicate that the extent to which the walls of the appendix are involved varies widely. In most instances, the infection occurs through the mucosa. It is probable that the primary infection, with ulceration in this situation — described by Aschoff[6] — is not really the beginning of the disease.

The lymphatic apparatus of the gut is invaded by bacteria through an intact mucosa in the course of a number of infections (enteritis, tuberculosis, typhoid, etc.), and it is certain that the same phenomenon occurs in the appendix. There is no reason why the appendix should be a law unto itself and not act in accord with the rest of the gut. The mucosa of the extirpated appendix often presents a picture of catarrhal inflammation very similar to that observed in connection with a similar condition in other portions of the gut.

These acute catarrhal conditions of the appendix, with or without involvement of the peritoneum, often end in complete recovery; the secretion is absorbed and the exfoliated epithelium regenerated; subsequent obliteration of the lumen is rare. Occasionally a defect in the mucosa is left after this form of infection; healing takes place by granulation. Deformation of the appendix (atresia and stenosis) depends upon whether the muscularis and subserous tissues are involved.

If the process at this stage does not recede, but extends, a phlegmonous inflammation is developed and *appendicitis gangrenosa,* with partial or extensive gangrene, invades the mucosa, and later the wall of the appendix, which it perforates. Occasionally this form of appendicitis heals spontaneously; in this event, the appendix is permanently obliterated by scar tissue formation.

There are, of course, various transitional forms between the simple catarrhal and the gangrenous. An important class is that of circular constriction, which completely occludes the lumen and leads to the formation of a cystic appendix (hydrops processus vermiformis).

When the inflammation is attended with excessive secretion, empyema of the appendix develops; this may not be accompanied by grave changes in the wall of the part. In these instances, a restricted portion of the appendix, or several multilocular areas, may be involved.

In the acute gangrenous perforating form of the disease, the organ

FIG. 915.— VARIOUS FORMS OF APPENDICITIS.

A, Catarrhal appendicitis. The process has not quite receded. The organ is much thickened. *B*, Subacute appendicitis. The entire organ is much thickened. *C*, Gangrenous appendicitis. Perforation has occurred near the tip. *D*, Gangrenous appendix laid open. A concretion is located within the lumen of the organ.

is red and covered with fibrinous deposits. Frequently, however, certain areas are green or black in color, the outcome of necrosis. The perforation may be the size of a pin point or the entire appendix may gape widely. An enterolith often lies at the site of the perforation, and, in many instances, escapes through it. How much of the attendant thrombophlebitis is primary is not certain.

In a general way, the phlegmonous abscessing and the hemorrhagic necrosing forms are differentiated. However, these sometimes merge into each other.

In perforation and gangrene the infectious mass may be discharged into the free peritoneum or into the vicinity of the appendix, where a serofibrinous exudate has already been formed. In other instances, residual adhesions may isolate the area of infection. Thus, an extending or a *circumscribed* form of *peritonitis* may be developed, the intensity of which depends upon the promptness and size of the perforation, but more especially upon the virulence of the invading excitants.

In the course of the inflammation the appendix may become attached to other organs: for instance, a loop of ileum or the cecum, and the perforation may occur into one of these. At other times, not often, the discharge of an enterolith may be followed by healing and an interval operation may not disclose the point of egress of the former, but may find it embedded in the contiguous adhesions. The gangrenous process may extend to the cecum; this complicates ablation of the diseased organ very much and is often followed by fistula formation.

Despite rather extensive gangrene and peritonitis, spontaneous healing is possible.

Peritonitis is caused by extension of the process from the appendix to the peritoneum and, in varying degrees, almost always complicates acute appendicitis. Its pathology does not differ from that already taken up in connection with inflammations of serous membranes (p. II, chap. x). At this time it is, however, advantageous to mention the peculiarities of *circumscribed peritonitis* developing in connection with appendicitis, the outcome of its varying position in the abdomen, i. e., in front, outward, behind, inward and in the pelvis. In these instances the process is similar to that already taken up in connection with circumscribed peritonitis. The additional and most important localizations are:

First, *subphrenic abscess* which usually invades the right subphrenic

fossa, but may, as already indicated (p. 1900), extend to the left and, by the lymph spaces, gain access to the pleural cavity.

The second localization occurs in the *retrovesical* space (in women, in the pouch of Douglas) and is the most frequent. In women the process possesses the characteristics of a postuterine abscess, and, in men, points in the ischiorectal fossa of either side, usually the right.

At times an appendicular abscess points anteriorly, either under Poupart's ligament, over the cecum, or, occasionally, at the navel or lumbar region. In this event, the skin is extensively undermined and must be widely opened before the process is exposed.

Spontaneous rupture of an appendicular abscess may be followed by healing; however, this does not protect the patient against recurrent attacks of appendicitis. Rupture of an abscess into the bladder is uncommon; its occurrence is attended by the passage of pus and gas with the urine. Perforations into the distal organs, such as the gallbladder, the uterus, etc., are attended with the formation of extensive intestinal adhesions. Most serious of all is extension of the inflammatory process to large vessels, which, when they are eroded, leads to fatal bleeding. Involvement of the walls of the veins may be preceded by thrombosis; subsequent embolism is then, of course, often followed by pyemia. In another class of cases, the thrombosis manifests itself in phlebitis of either side.

Chronic Appendicitis.— Chronic appendicitis is characterized by adherence of the appendix to contiguous parts and often by swelling of the lymph nodes. The lymphatic follicles of the appendix are almost always enlarged; its walls are thickened and its lumen more or less stenosed. In this way the appendix is made to consist of hollow spaces, separated from one another, and filled with secretion. Enteroliths are often found in a space of this sort. Frequently, the appendix is kinked or twisted — the main cause for the retention of the contents. These kinkings are often due to anatomical peculiarities; however, there is no doubt as to the causative relationship of chronic inflammations in this connection. In this class of cases, the cecum in the region of the appendix is also more or less thickened and the contiguous ileum is similarly affected, while, in not a few instances, the ileocecal region is embedded in veil-like adhesions, especially in the vicinity of the valve of Bauhin; these adhesions cause changes in the relationship that the cecum and the ileum bear toward each other. The underlying causative conditions in this class of cases is, no doubt, a variable one, i. e., at one time it may be a typhocolitis, with or with-

out appendicitis, at another, appendicitis with or without typhocolitis.

Tuberculous Appendicitis.— Isolated tuberculosis of the appendix is very rare; it usually occurs in connection with tuberculosis of the cecum (p. 2001). The process may be of the ulcerative, the hyperplastic, or the peritoneal type. It rarely perforates into the peritoneum, but occasionally forms an abscess which perforates into the gut. Isolated tuberculosis of the appendix is usually accidentally discovered during an operation for relief of chronic appendicitis.

Actinomycosis of the appendix is attended with the pathological changes characteristic of this disease in other portions of the intestine. It is not recognizable as such until the extirpated specimen is subjected to microscopical examination.

The appendix is occasionally found in a *hernial sac*. When strangulation occurs it may be difficult to decide whether the resultant necrosis is due to the strangulation or whether a herniated appendix has become infected. The presence of enteroliths and the coëxistence of more or less peritonitis argues for the latter.

Tumors of the appendix are uncommon. Carcinoma has been most frequently found and probably is not so rare as has been believed. Over one hundred cases are reported in the literature. The growth is recognized only upon microscopic examination. It appears usually at the distal portion of the appendix in the form of a club shaped tumorous enlargement. Most of the tumors are adenocarcinomata (Luce[7]). Sonnenburg[5] reports a case of lymphosarcoma of the appendix.

Clinical Course of Appendicitis — Symptoms in the First Forty-eight Hours.— Acute appendicitis may appear without previous warning. In most cases, however, inquiry elicits the previous existence of mild disturbances, such as loss of appetite, etc., or the general discomfort common to all infectious diseases. There are, of course, cases in which a perfectly normal appendix becomes acutely inflamed. However, the rule is, that an acute attack is usually preceded by more or less definite disturbance in the right iliac fossa. A certain number of cases are preceded by bladder symptoms, especially in women.

The forerunner of acute appendicitis, *colica appendicularis*, occurs in the majority of cases. It is caused by the spasmodic expulsion of retained secretion. The colicky pain begins suddenly and is usually localized in the right iliac fossa; it may last for a few moments or may persist for several hours. The attacks are followed by vomiting and, in children, by constipation with slight abdominal distention.

Whether acute appendicitis is preceded by colic or not, the impression is that the disease develops in a healthy person. The clinical picture begins with *pain,* which is very likely to be severe and is usually attended with vomiting. The patient is ill enough to go to bed. The intensity of the pain varies with the changes in the appendix and the amount of retained secretion. As soon as the peritoneum is invaded the pain becomes very severe and is likely to be diffuse. Perforation is often attended with sufficient pain to cause collapse. The pain may at first be referred to the epigastrium or navel and may gradually localize in the right iliac region.

Leukocytosis is a valuable corroborative sign of appendicitis. Murphy[2] says "The rôle of the polynuclear cell is excellently estimated by Albrecht.[8] The percentage of polynuclear cells is an indication of the severity of the infection, but not of degree of destruction; a high percentage does not denote a bad prognosis, so long as the absolute number of white cells is correspondingly high. A fall in the absolute numbers of polynuclears with a coincident decrease of the total percentage of leukocytes, shows a decline in the infection; a sudden fall in the absolute numbers of polynuclears, with a very small decline, or perhaps a rise in the percentage of white cells, is of unfavorable prognostic import, since it shows that the production of the polynuclears does not keep pace with their consumption, indicating an exhaustion of the blood forming elements for defensive purposes. A very unfavorable sign is a sudden fall in the total number of leukocytes. Beginning recovery is manifested by a rise in the absolute number of mononuclear and eosinophilic leukocytes, even to or above the normal. We have never found leukopenia with acute infections of the appendix."

Of the local symptoms, *tenderness,* together with more or less diffuse pain in the abdomen, is the most characteristic. The local tenderness often corresponds to McBurney's point, but varies, of course, with the position of the appendix. At times tenderness may be elicited per rectum. The greater the tenderness the more marked is *muscular rigidity;* the latter is strongly indicative of appendicitis. The presence of these two symptoms, in the absence of others, argues strongly for the employment of operative measures. In the first few hours of the disease percussion and auscultation are negative.

In the light, so-called, catarrhal attacks the local and constitutional symptoms are very moderate in degree. Fever is slight, the pulse not very much accelerated, leukocytosis absent; the attack recedes in

twenty-four hours. This class of cases is difficult to differentiate from catarrhal typhlocolitis, probably because the appendicular symptoms are really a part of the former, and both soon bow to cleansing of the bowel.

It is unfortunate that acute attacks of appendicitis are likely to follow an erratic course, one which cannot be foreseen. For this reason, the temperature, pulse, and, especially the blood picture, must be studied repeatedly and at brief intervals.

The *diagnosis* is simple enough in cases in which the temperature, pulse rate and the blood picture carry out an accord with the local disturbances. In these cases, beginning peritonitis is also evident; the abdomen is somewhat distended, the urine is scanty, and the bowel constipated. As the case progresses, vomiting begins, the temperature rises, the pulse rate is progressively increased, leukocytosis is greater, and the patient's general condition indicates the presence of a grave disease.

Symptoms of Acute Appendicitis After Forty-eight Hours and Its Subsequent Course.— The farther course of acute appendicitis (when not operated upon) after forty-eight hours, leaves little to the imagination. Recession is attended with falling of the temperature, pulse rate, and leukocytosis; the normal is reached by the fifth or sixth day. The tongue clears up, the general appearance of the patient improves, meteorism and tenderness disappear, and gas is expelled per rectum; if abdominal rigidity has been present this gradually fades. These are the cases in which the secretions retained in the appendix are expelled and the exudate absorbed. When this does not happen the symptoms of peritonitis gradually dominate the picture, i. e., either circumscribed or extending peritonitis develops. While pain is the most indicative symptom in this connection, it may be absent.

Even the development of *circumscribed peritonitis* does not preclude spontaneous healing of the disease. The symptoms of localized peritonitis consist in changes in the percussion note and increased resistance in the ileocecal region. In this form of localization the general symptoms rapidly decline, vomiting ceases and the abdomen becomes flat. The tender tumor is not the appendix, but is due to infiltration of the cecum and last portion of the ileum, and the exudate deposited around the appendix. In suppurative appendicitis the tissues in its vicinity are always the seat of inflammatory edema. A circumscribed abscess is the result of an encapsulated purulent peritonitis.

This description applies to the cases in which the appendix lies in its normal position. When the appendix is turned upward or lies behind the cecum, circumscribed peritonitis is likely to be retrocecal and extend up to the diaphragm without involving the general peritoneum. As a rule, resistance is masked by the interposition of loops of small gut. Tenderness is most marked in the lumbar region, and the leg is flexed. The latter position is more indicative of abscess located on the ileopsoas muscle. Collections of pus in the true pelvis cause bladder disturbances (tenesmus) and may often be felt through the rectum or pouch of Douglas.

Rupture of the appendix and the sudden entrance of a large quantity of pus into the general peritoneum is attended with an immediate exacerbation of all the symptoms. As soon as the shock is over the clinical picture of general peritonitis (already described, p. 1890) rapidly develops.

Acute general peritonitis developing in connection with appendicitis of virulent character is very likely to be fatal. In fulminating cases, leukopenia is often presented. In the less virulent, multiple encapsulated abscesses may be formed. Abscess formation is heralded by a sudden increase in leukocytosis, followed by increase in pulse rate and temperature. The exudate may often be discovered by physical examination. The rupture of localized collections of pus of this sort is followed by a renewed attack of general peritonitis.

Abscesses formed in the region of the appendix may rupture into the gut, the cecum or the ileum, the bladder, the uterus, the vagina, or through the skin. These events are often followed by incomplete healing of the process and the development of new complications. Of the various abscesses, the subphrenic is the most serious.

Additional complications of acute appendicitis consist in *ileus,* which may be a part of general peritonitis or may develop as the result of kinking of coalescing loops of gut. Ileus is often preceded by singultus.

The danger of *thrombosis and embolism* in connection with acute appendicitis is well known. Patients afflicted with perityphlitis are always thrombus carriers and but little additional infection is necessary to develop an acute attack near the right iliac fossa or remote from it. The embolism need not necessarily be septic; slight secondary embolic inflammatory processes frequently occur, even after operations during the so-called free interval.

The *clinical picture* of acute appendicitis after the first forty-eight

hours may be said to be that of peritonitis and is dependent upon whether this is encapsulated or not. A complication of the first forty-eight hours — that of thrombophlebitis — must be mentioned; it is usually fatal.

Acute appendicitis during pregnancy claims a high mortality. Only an early diagnosis (not by any means easy) and immediate operation obviates a fatal outcome. Premature expulsion of the fetus is not by any means a certainty.

The Symptoms of Chronic Appendicitis.— Chronic appendicitis appears in two forms, the one occurring sequentially to an acute attack or the result of repeated attacks of moderate degree; and the other, which is insidious in its onset and never develops into an acute attack.

The form following an acute attack is characterized by the peculiarity that, after subsidence of the acute symptoms, the *right iliac fossa is never entirely free from pain and tenderness* and that from time to time acute exacerbations of the process develop. In the second form the clinical manifestations are not so clear, the main symptoms consisting in obstipation, tenderness over McBurney's point, and slight distention of the cecum. If the inflammation is attended with the formation of adhesions, this may be demonstrable by the Röntgenogram.

When tenderness is clearly defined (after repeated examinations) and the patient experiences pain in this region for a protracted period of time, the surgeon is justified in regarding the case as "suspicious."

It is of course true that the symptoms ascribed to chronic appendicitis are also provoked by typhlocolitis. Cecum mobile, so-called, is of itself not responsible for appendicitis. Differentiation with regard to the various processes responsible for pain and discomfort in the right iliac fossa requires careful study and patient observation, and the intelligent application of the various available diagnostic measures. A positive diagnostic conclusion is not a little hampered by the fact that the appendix is often responsible for reflex gastric symptoms.

In the *differential diagnosis* the blood findings are of great value; in the first week of *typhoid fever*, leukopenia excludes appendicitis. While *pneumonitis* is also attended with leukocytosis, the incongruity of the local symptoms as compared to the constitutional disturbance should prevent error in this connection.

Since *acute and chronic colitis* has come into the domain of the sur-

geon, and the advent of the Röntgenographic examinations have given more or less definite information in this connection, it has become apparent that the appendix is often involved as a part of inflammatory processes attacking the gastrointestinal canal, and this non-resisting blind pouch may heal together with the disseminated inflammation, or it may become gangrenous and perforate when the general process seems to be tending toward recession. This is especially true with regard to acute circumscribed colitis; however, the latter is not attended with peritoneal invasion and the blood picture does not reveal abnormalities. *Typhilitis occurs in appendectomized persons.*

In searching for the cause of pain and tenderness in the right iliac fossa, a number of possibilities must be taken into account, and their distinguishing manifestations carefully sought for. These include *invagination, internal hernia,* inflammation of an *undescended testicle, acute tuberculous peritonitis, cholecystitis, ureteritis, gastric ulcer,* and, in the female, *adnexal disease.* With regard to *chronic appendicitis,* the chronic inflammatory diseases of the biliary passages, the gut, the female adnexa, and the stomach must be especially taken into account. When a *tumor* is present, growths originating from the female pelvic organs (twisted ovarian cyst) or cecum (especially carcinoma and tuberculosis) must be borne in mind. In this regard the Röntgenographic findings are of great diagnostic importance.

So many factors enter into the *prognosis* that a conclusion based on statistical analysis is of little value. In all cases, the persistence of acute symptoms for forty-eight hours indicates a menacing condition of affairs. The realization of this and the almost universal employment of the early operation has markedly reduced the mortality. As in all infectious processes, the outcome depends upon the virulence of the invading horde of excitants and the resistance of the host. Basing the prognosis on the local process, it becomes increasingly unfavorable as the evidences of peritonitis and its attendant ileus becomes patent. The final outcome is the result of the constitutional effect of the latter.

With regard to the *treatment of appendicitis* it is permissible to state that operative measures are indicated as soon as the diagnosis is established. Difficulty with regard to the proper procedure is influenced only by the latter consideration. In doubtful cases the prompt appearance of evidences of recession of the process justifies awaiting substantiation of the diagnosis; on the other hand, increasingly menacing symptoms in the doubtful cases may be properly regarded as indicating the operation. It is true that, in not a few instances, cases

which present a clinical picture suspicious of appendicitis clear up entirely after a dose of castor oil; however, these are cases of typhlocolitis and do not present the blood picture common to *appendicitis with peritoneal involvement.* The technic of appendectomy, the treatment of appendicular abscess (circumscribed suppurative peritonitis), and that of general peritonitis and its attendant ileus, have already been taken up in chapter iii of this part. In this regard the conditions mentioned do not present regional peculiarities; a special discussion of them leads only to confusion.

The formation of a fistula, either spontaneous or postoperative, is of two kinds, those discharging *pus,* and the *fecal.* Most of the fistulae of both classes heal spontaneously; failing in this, the employment of the measures described above (p. 1955) is indicated.

Other than the bibliographical references mentioned, those quoted by Murphy[2] may be studied with advantage.

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CHAPTER XII

HERNIA

General Considerations.— A HERNIA MAY BE DEFINED AS A PROTRUSION OF ONE OR MORE OF THE ABDOMINAL VISCERA THROUGH A NORMAL OR AN ABNORMAL OPENING. The chief cause of hernia of this sort is a congenital defect, while the immediate exciting cause plays a minor part (Coley[1]).

A hernia is composed of three parts, (1) the sac; (2) the covering of the sac; (3) the contents of the sac. Most writers divide hernia into *congenital* and *acquired.* Inasmuch as the only means of determining that a sac is preformed is by the fact that it communicates with the tunica vaginalis, it has been customary to class as congenital only those showing this anatomical peculiarity. It is probable that in the vast majority of inguinal hernias in the male, and practically all in the female, the sac is preformed, i. e., the opening in the funicular process persists, even though the hernia may not develop until adult life. For extended discussion in this connection see Coley[1] and Russel.[2]

From an anatomical standpoint hernia is divisible into (1) *inguinal;* (2) *umbilical;* (3) *femoral;* (4) *ventral,* in the order of frequency stated. The rarer forms are obturator, ischiatic, pelvic, and lumbar.

Clinically, hernia is classified as *reducible, irreducible, inflamed,* and *strangulated.*

Reducible hernia is by far the most frequent form; the contents of the hernial sac may return to its normal environment either spontaneously, when the recumbent position is assumed, or as the result of manipulation. As a rule, all hernias are reducible early in their history.

The Hernial Sac.— The *hernial sac* always consists of peritoneum, which either completely or incompletely covers the contents. In the *congenital* variety the sac is a preformed pouch or diverticulum of peritoneum, which is empty, until by some unusual effort or accident some of the abdominal viscera are forced into it. In the *acquired*

variety, the best examples of which are seen in the direct inguinal and ventral hernias, the parietal peritoneum is forced through a congenital defect in the abdominal wall. Under certain conditions the hernial sac may not inclose the entire projection; this is seen in protrusions of the cecum, sigmoid, and other organs not entirely covered by peritoneum. In these instances, the dislodged peritoneum covers only the anterior surface of the hernial projection — sliding hernia (Leriche[3]).

The rarer internal hernias, such as the diaphragmatic, are not usually covered by peritoneum. In ventral hernia following celiotomy, especially when drainage has been employed, the gradually thinning scar permits egress of the abdominal contents, which may be covered in part by peritoneum or not at all.

A hernial sac is described as possessing a *mouth*, a *neck*, and a *body*. The form of the body varies somewhat, being either club shaped (with its base pointed centrally), cylindrical, bag shaped or irregular; occasionally, one or more diverticula project from the side of the sac (multilocular sac). Not infrequently the sac possesses areas of constriction, probably because one sector is more distensible than another; or, in congenital hernias, the constrictions correspond to partial obliteration of a funicular process; or, what is no doubt often the case, a contracted neck is displaced downward by an increase in the size of the hernia.

In recent hernias the inner surface of the sac possesses the characteristics of peritoneum; in older cases the color is slate gray, the serosa is studded with areas of thickening and is discolored from old hemorrhages. Occasionally, the sac is cartilaginous or, in part, calcified. In others, the sac is the site of chronic inflammation, which results in *cohesion* of its surfaces or in the formation of *adhesions* between the sac and the contents. When a hernia is persistently held back by apparatus, the inflammation may result in total obliteration of the sac. In other cases, it is responsible for insular collections of serous fluid, which take on the form of *cysts*. At times, a new hernial sac sinks into one of these cysts (Cooper's *encysted hernia*).

A protrusion of parietal peritoneum may occur without an attendant prolapse of abdominal contents, i. e., a funicular process or the diverticulum Nuckii may remain patent and no hernia descend into it. In addition to this, a number of protrusions of the peritoneum are simply filled with lipomata; these are found in the inguinal and crural regions.

The Hernial Contents.— A hernial sac becomes a true hernia only

when abdominal contents enter it. Almost all the abdominal organs have been found in a hernial sac. When most of the abdominal organs are prolapsed the term *eventration* is used.

The *gut and omentum* are most frequently herniated. As a rule, the same portion of gut wanders in and out of the sac; finally, it undergoes changes which lessen its nomadic trend, but, like all vagrants, shows a tendency to remain in the abnormal position.

In the majority of cases, the upper portion of the ileum, where the mesentery is the longest, constitutes the primary contents of a hernia. As a rule, a loop, together with its mesentery, herniates; at times, three or four loops are present. Not infrequently, the hernial contents consists of *only a part of a given sector of the gut* or of a diverticulum. These hernias may consist of:

1. A *congenital* (Meckel's) *diverticulum* (the genuine hernia Littré[4]).

2. A *false diverticulum,* i. e., a bulging of the wall of the mucosa of the gut through a defect in the muscularis; these are often multiple.

3. *Hernia of the intestinal wall* (partial hernia, lateral hernia). The hernial contents consists of only a part of the wall of the gut — usually its free border (opposite the mesentery). If this persists for a long time, the herniated part resembles a diverticulum; it may become strangulated.

Occasionally, the *transverse colon* has a long mesentery and is herniated; this is also true with respect to the sigmoid and cecum, both of which, being partially covered with peritoneum, form *sliding hernias.*

After the gut, the omentum is oftenest found in a hernia. In children the omentum is very small, but in the adult it is likely to cover the entire gut, and often possesses projections which are readily prolapsed into a hernial sac. Most umbilical, and many ventral, hernias contain omentum.

With regard to the frequency with which the various portions of the gut, with or without omentum, form the hernial contents in strangulated hernias subjected to operations, Hilgenreiner[5] reports the following: Small intestine 85 per cent; small intestine alone 60 per cent (wall of the gut 5.3 per cent); small intestine with omentum 24 per cent; small and large intestine 2 per cent; small and large intestine and omentum 0.5 per cent; omentum alone 9.6 per cent; omentum and colon 2 per cent; omentum with gut 35.6 per cent; colon 7 per cent — alone 2.7 per cent.

The *bladder* is not rarely found in a hernia; one hundred and

eighty cases have been collected by Brunner.[6] The prolapsed bladder may be completely or incompletely covered by peritoneum; the latter is the rule.

Of the *female organs of generation*, the tube and ovary are most often herniated; however, the uterus has also been found in a hernial sac.

The *liver* may be herniated through the diaphragm, although it has also protruded through the navel.

Rose[7] reports finding the pancreas in an umbilical hernia.

Hernia adiposae deserves special mention. The fatty tissue, in the form of a lipoma, is derived from the subcutaneous tissue and may protrude through any of the openings where hernia occurs. This condition, unless borne in mind, may lead to confusion during the operation, especially when associated with a true hernia.

Accessory Hernial Coverings.— The *outer coverings of a hernia* lying between the sac and the skin vary widely. In not a few hernias, especially the umbilical, the sac lies immediately beneath the skin and may be adherent to it; on the other hand, the majority of hernias are covered by layers of tissue (fascial, fat, aponeuroses, and muscles) lying over the course the protrusion follows in its egress. For the anatomist these layers possess great theoretical importance, but the surgeon who expects to find them during the operation will be much disappointed to find the lines of demarcation in this connection obliterated by pathological changes. As a rule, the transversalis fascia can always be found, and is, therefore, called the *fascia propria*.

The Symptoms and Diagnosis of Hernia.— In a general way one may say that the diagnosis of hernia is certain WHEN THE CONTENTS OF AN ABDOMINAL PROTRUSION MAY BE RETURNED TO THE ABDOMINAL CAVITY, or when one is able TO ESTABLISH WITH CERTAINTY THAT THE CONTENTS OF A SWELLING CONSISTS OF AN ORGAN WHICH IS LOCATED IN THE ABDOMEN. When reposition succeeds it is often possible to lift a fold of skin and, by invaginating it with the finger, to determine the presence of a hernial port, and its length and direction. If the patient now coughs, the finger feels the impact of the returned viscus (*impulse on coughing*), and when the finger is withdrawn the gut is usually again protruded.

When the tumor is not reducible, its CONNECTION WITH THE ABDOMINAL CAVITY MUST BE ESTABLISHED. This is best accomplished by observing the disturbances in the normal outline of the body, especially with regard to the sharply defined line corresponding to Poupart's

ligament. The surgeon searches for the pedicle of the tumor and observes the extent to which it is movable, its form, and its consistence. It is important to note the variations in size which the tumor undergoes at different periods of the day and in accord with the position of the patient (supine, seated, erect, etc.).

It is, of course, exceedingly helpful to determine that the contents of the tumor consists of an organ normally located in the abdomen.

Intestine is recognized by its symmetrical rounded outline and elastic consistence, the latter varying with the pressure that the patient makes with the abdominal muscles. In large hernias, peristalsis is not infrequently visible. Intestinal rumbling, spontaneous or provoked by efforts at reduction, is an important diagnostic sign.

The *omentum* presents an irregular nodular mass, which is characteristic. If gut is also present, this "goes back" first; the omentum is not always readily reduced. The latter may be confused with hypertrophied appendices epiploicae or a much thickened sac.

Identification of other organs is not always possible. The presence of a portion of the female adnexa may be assumed when vaginal manipulation of the uterus influences the outline of the hernial protrusion.

When the *bladder* is herniated, only a portion is protruded. Pressure on the latter may provoke a desire to urinate. Retention of the urine causes the prolapsed portion to enlarge; the latter disappears when the urine is voided.

The Influence of Hernia on the General Condition.— Many persons are afflicted with large hernias without being aware of the condition until it is accidentally discovered or a complication arises. However, in most instances, a hernia makes its presence known in some way. Many patients complain of boring or tearing pain in the lower abdomen, digestive disturbances, and even occasional attacks of vomiting. Observing patients note that the feeling of discomfort moderates when the prone position is assumed; this should arouse suspicion of a hernia. A properly fitting truss usually relieves all the symptoms. Patients with fully developed hernias, and especially those which cannot be satisfactorily retained by a truss, are almost invariably constipated.

The Treatment of Hernia — Prophylaxis — Trusses.— The prophylactic treatment of hernia has a useful field in the *first years* of life. All children should be examined for hernia, and causative factors, such as a tight phimosis, constipation, persistent cough, or unnecessary crying, corrected.

The *palliative* treatment of hernia consists in treating the disturbances coincident to it without correcting the hernia itself. This includes *reposition* and *retention* of the protrusion.

An irreducible hernia should never be subjected to the pressure of a retention apparatus — a truss. In cases in which the gut is reducible and the omentum remains prolapsed, it is, at times, for special reasons, permissible to employ a truss with a hollow pad.

Many reducible hernias return to their normal environment when the patient assumes the recumbent position. In other cases very simple manipulations accomplish the desired end.

Reposition is made easier by preliminary emptying of the bowel and bladder, and by relaxing the abdominal muscles. The latter is best

FIG. 916.— DE GARMO-HOOD TRUSS: GERMAN SILVER SPRING, HARD RUBBER COVER. Applied to right oblique inguinal hernia (De Garmo).

accomplished by elevating the thigh and slightly erecting the trunk; the head is best lowered. Reposition of large hernias is, occasionally, attended with a feeling of discomfort and tension in the abdomen, nausea, vomiting, and syncope, which persist until the hernia is again allowed to prolapse. In these cases the patient is *prepared for the reduction* by restriction of diet, brisk catharsis, and the maintenance of the recumbent position for several days. Each day a careful effort is made to effect the reduction until it finally succeeds. This is immediately followed by the application of an appropriate truss.

Retention of a hernia by means of a truss was taken out of the hands of charlatanism by Camper[8] (1771). It is, of course, impos-

sible to enter into a detailed discussion of the subject here. However, it is permissible to suggest to the practitioner that the fitting of a truss does not require "genius;" on the contrary, the working out of the principle involved is within the grasp of the normal mind.

De Garmo[9] says, in substance, *Truss fitting* consists of obtaining the measure and shape of the patient, selecting the truss suited to the case, shaping the spring, and its application to the patient. A patient who obtains a truss from the manufacturer is fitted "by accident."

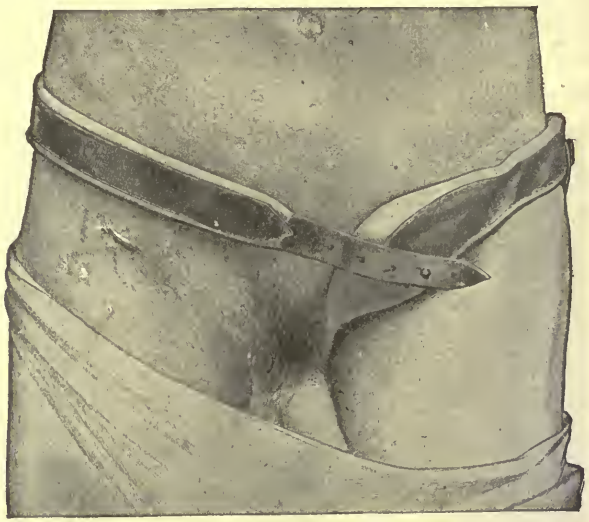

FIG. 917.—ENGLISH "RAT-TAIL" TRUSS (De Garmo).

Each truss should be fitted to the form of the patient who is to wear it. For the purpose, the surgeon outlines on paper the portion of the body to be fitted, obtaining this first by the lead tape method. The pliable lead tape is made to conform to the body and its outline is readily transferred to a sheet of paper; the truss spring is made to conform to the diagram. The adjustment of the spring should be such that the hernia is retained without causing pain. Of the various kinds of springs used, those of tempered steel or German silver are probably best suited to the purpose. A properly fitted truss does not require perineal or shoulder straps. Large hernias are well controlled by the English rattail truss shown in Fig. 917.

The *pad* which occludes the hernial opening should be moderately convex and oblong in shape; there is little doubt but that those made

of hard rubber or celluloid are suitable for most cases. The connection between the spring and pad should be so arranged that adjustments in this connection are feasible; a locking ball and socket joint is universally used for the purpose.

In all cases the apparatus should be worn next to the skin. It should be kept *in situ* except when the patient is in bed. In cases of large hernias, a light truss should also be worn at night.

In a general way, the employment of a truss is a palliative measure. However, there is no doubt that *occasionally* the *prolonged use of a truss is followed by a radical cure,* especially in children.

The Radical Treatment of Hernia.—The radical healing of hernia may be accomplished by the following means:

1. *Use of a truss,* which is, however, applicable only in young persons and is successful only when the hernia is kept fully reduced so that physiological closure of the aperture may take place.

2. *Obliteration of the hernial port by the production of an adhesive inflammation.* This consists in the injection of tincture of iodin, etc. The method does not often succeed and is no less dangerous than the open operation.

3. The *production of a pelotte by the injection of coagulating substances.* Gersuney[10] uses vaselin for the purpose, and Eckstein[11] paraffin. The method succeeds in small hernias in children; it may be employed in the adult when operative measures are contraindicated.

4. *Operative measures which close the sac and the hernial port* (Kallionzis[12] gives an excellent review of the literature in this connection). The principle of all herniotomy operations is based on *extirpation of the sac* and *narrowing the hernial port.*

The technic of the various procedures is taken up with the discussion of special hernias; at this time only the general principles are discussed.

The important requisites of every herniotomy are: (1) the *neck of the sac must be deligated sufficiently high, so that no funnel shaped protrusion is left* in the vicinity of the port of exit; (2) the *port of exit must be closed as firmly as is possible;* (3) *healing by primary union* is essential; (4) avoidance of complicated methods; (5) full exposure of the operative field.

The radical operation is possible only when the herniated structures are completely reducible or are susceptible of removal. Adhesions between the contents and the sac must be divided. In certain instances,

it is permissible to reduce a portion of the sac firmly adherent to the contents with the latter.

When the omentum is adherent, the prolapsed portion is best amputated after sectional ligature of the stump. Extensive resection of the omentum involves danger of the formation of inflammatory tumors and subsequent ileus (Braun[13]).

When the herniated parts are readily reducible, *opening the sac* is not necessarily indicated; however, when this is cautiously executed the assurance that all of the contents is returned is gratifying. The section should not be carried high enough to involve the risk of incomplete closure of the mouth of the sac.

After the sac is entirely isolated, it is ligated sufficiently high to obviate the formation of a central funnel shaped depression. If the neck of the sac is wide, it is best to close it with a *purse string suture.* Closure of the sac is particularly difficult in cases of sliding hernia. In all instances, ligature of the sac is best accomplished by transfixion.

Hemostasis must be complete; failure to secure this leads to serious complications. Drainage of the superficial wound is necessary only when there has been much trauma to the tissues during the manipulations.

Plastic closure of hernial openings is employed when firm approximation of the edge of the hernial port is not feasible. Autogenous or exogenous methods have been used with varying success. In 1890, Trendelenburg[14] transplanted a portion of the humerus, and, later, turned an osteoplastic flap upward from the pubis into a hernial defect. Since then the tendency has been to use free transplants from various portions of the body; of these, a portion of the fascia lata (Kirschner[15]) and of the periosteum (Henschen[16]) have been used with considerable success. In all cases the hernial port should be closed as far as is possible by suture and the transplant used as a reinforcement.

The use of heteroplastic material, such as silver wire, has not proved of permanent service (Witzel[17]).

Accidents of Hernia.— The most important accidents of hernia are obstruction, inflammation, and strangulation.

Obstruction.— Obstruction of the herniated gut is due to the gradual accumulation of feces, which leads to complete stasis.

This is most likely to occur in a large irreducible hernia in aged persons and is ascribed to paralysis of the gut, i. e., the previously hypertrophied musculature degenerates and is incapable of performing its function.

Most patients afflicted in this way are, for a long time, subject to a certain degree of discomfort and are likely to be unappreciative of the impending disaster. The hernia is not tender, nor red, nor is there much general disturbance. However, in the end, the abdomen becomes distended, nausea and vomiting follow, and, finally, the picture of intestinal obstruction develops.

The *diagnosis* is based on the history of gradual obstipation, the doughy feel of the hernial contents, and the absence of the symptoms of acute constriction. The picture, as a whole, closely resembles that of *obturation ileus.*

The *treatment* is that of fecal impaction in general. This succeeds best when the hernia is reducible. The use of cathartics is objectionable. Colonic irrigation is permissible. If constriction is suspected, small doses of opium may be administered. Gastric lavage is indicated. Failure to afford relief by the means stated justifies the employment of operative measures of relief.

Inflamed Hernia (Peritonitis Herniaria).— As the hernial sac and its contents are covered with peritoneum, its inflammation possesses all the characteristics of peritonitis.

The *mild forms* are due to *mechanical lesions,* such as a contusion, with or without bleeding, or are the outcome of prolonged truss pressure, or undue manipulations in an effort to accomplish reduction of the protrusion. Not infrequently the inflammation originates in a *twisted omental projection.* In other instances it is secondary to an affection of the prolapsed gut, such as ulcerative enteritis, tuberculosis, etc. The *appendix* is of special importance in this connection. A number of instances of appendicitis and typhlitis in connection with herniated ceci, followed by secondary *peritoneal phlegmon,* have been reported (Graser[18]).

Tuberculous infection of the hernial sac has been frequently observed. Extension of an inflammatory process, involving the peritoneum, to a hernial sac is, of course, readily understood; it occurs most often in connection with puerperal infection.

The inflammation possesses all of the varying characteristics discussed in connection with peritonitis (p. 1890).

Lesions of moderate degree may subside without producing marked symptoms. In the others, the picture does not vary from that presented by inflammation in general. It is important to note that the disturbances *do not include constipation;* on the contrary, diarrhea is the rule.

The *treatment* in the lighter cases consists in keeping the patient quiet and in the administration of a cathartic. In the severer cases, the sac must be freely incised. In this event, the surgeon must be prepared to meet a possible strangulation.

STRANGULATED HERNIA (INCARCERATED HERNIA).— STRANGULATION IS A TERM APPLIED TO THE CONSTRICTION OF THE GUT IN THE HERNIAL CANAL, WHICH MAKES IT IRREDUCIBLE, DAMAGES THE CIRCULATION IN THE PROTRUSION, AND, IF NOT RELIEVED, LEADS TO GANGRENE.

Strangulated hernia is divided into *elastic constriction* (*strangulation*) and so-called *fecal constriction* (*incarceration*).

The *mechanism of elastic strangulation* is comparatively simple. The intra-abdominal pressure forces a loop of gut through the hernial port by stretching the latter. As soon as the pressure moderates, the elasticity of the opening causes it to contract and the gut is constricted. This also happens when a hernia is already present, i. e., when either an additional portion of the old protrusion or a new loop of gut is forced into the sac.

If the constriction is of high degree the gut cannot free itself; nor does taxis relieve the situation; the remedy lies in division of the constriction. The elastic constriction not only confines the gut and interferes with the passage of feces, but also interferes with the circulation in the prolapsed part. A moderate degree of strangulation interferes with venous return and a more severe one also arrests the arterial flow of blood. In the severest forms gangrene is established in a few hours, the gut is empty, and the sac does not contain fluid (anemic gangrene).

Fecal constriction (*incarceration*) occurs with the coöperation of the gas and fluid contents of the gut. A sudden increase of pressure forces a large quantity of gas and feces into the herniated gut. When the pressure is released the previously movable herniated gut becomes immovable; despite the exercise of great pressure the contents cannot be moved in either direction. This happens when the hernial port is so wide that *the question of constriction need not be taken into account.*

The *mechanism of incarceration* is mysterious. Despite its apparent lack of analogy to elastic strangulation, a sharp line of demarcation between the two is not possible. In incarceration there is also a narrowing of the intestinal lumen and a disproportion between the hernial port and the contents, which ultimately leads to strangulation.

The construction usually occurs at a ring shaped narrowing in the hernial course. This may be found in the sac or in its surroundings,

even in one of the coverings of the sac, and, occasionally, multiple constrictions are present.

Extended study of the mechanism of fecal constriction has been made by Roser,[19] Busch,[20] and Kocher.[21] There has not been anything added in recent years that does other than support the conclusions of these observers. Coley[1] quotes Zwalenburg as substantiating the theory of Kocher by ingenious observations made in 1915. Kocher[21] seems to have shown that the determining factor consists, not in filling the gut, but in its sudden distention above the ring, so that the mucosa and muscularis is forced into the constriction and obstructs the lumen of the gut.

In addition to this, kinking also plays a part in the mechanism; according to Chassignac,[22] the kinking becomes more marked as the herniated gut is more distended. Not a few authorities consider that the backward drag of the mesentery causes it to act as a wedge (apex toward the abdomen) which also compresses the gut.

Aside from the mechanical conditions mentioned, the *circulatory changes* coincident to constriction are exceedingly important. As already stated, these vary from a moderate degree of venous stasis to complete arterial occlusion; however, in the absence of all constriction, *distention of the gut* with gas and fluid also leads to grave disturbances in this connection. Distention of the gut is always attended with venous hyperemia, which ultimately leads to hemorrhagic infiltration of the wall of the gut and bloody effusion into its lumen. The stasis thus produced is also accompanied by *paralysis of the musculature* of the gut. It is readily conceivable how, even though the hernial port is originally ample in size, the gradually distending and infiltrating herniated gut would soon produce a condition of affairs ultimately leading to constriction, and that this, in turn, results in *gangrene*.

The absence of free anastomoses between the blood vessels of the gut explains the lack of resistance of the latter, which rapidly succumb to the ever present bacterial flora of the contents, and *gangrene*, together with putrefactive infection, promptly develops.

Hernial constriction of the gut is almost always attended with the transudation of fluid (*hernial fluid*), the quantity of which is proportionate to the degree of the constriction and the length of time it has persisted. The fluid is at first clear, amber colored, or serous in character, but soon becomes bloody and often contains flakes of fibrin. Gradually the addition of leukocytes clouds the transudate, which ulti-

mately becomes foul. When the gut is gangrenous, the entire sac is filled with a thin putrid fecal fluid and gas.

The question as to whether the hernial fluid has been invaded by bacteria has been carefully studied. Recent observations would indicate that these pests of the surgical field permeate the bowel and may

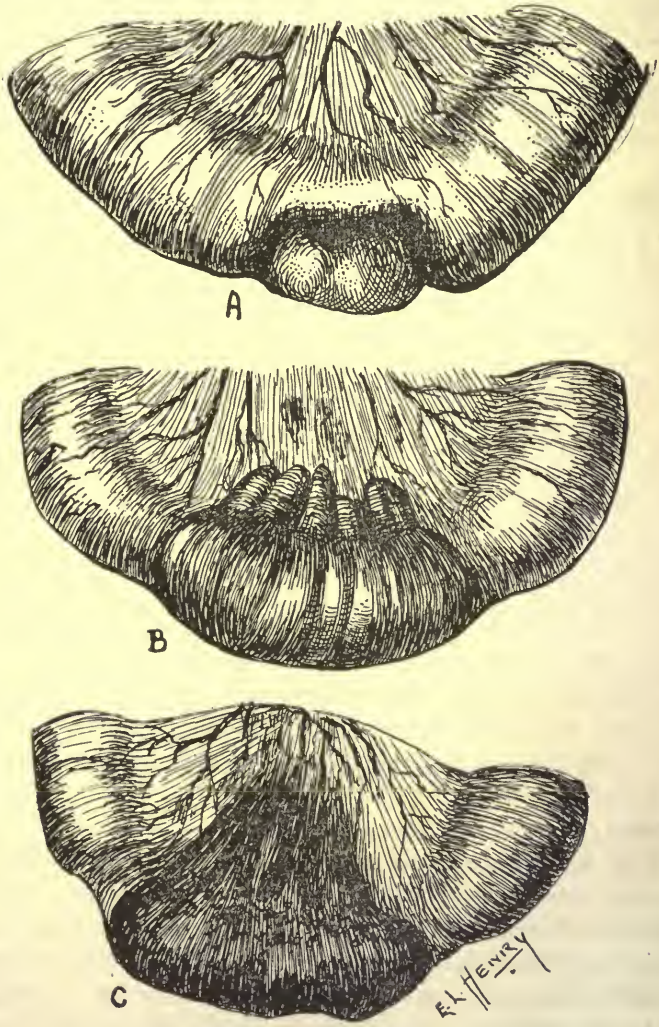

FIG. 918.— VARIOUS DEGREES OF STRANGULATED GUT.

A, Hernia of intestinal wall. *B*, Hernia of entire diameter of gut. *C*, Hernia of gut and mesentery.

be demonstrated in the fluid when the epithelium of the latter is damaged, which occurs when the constriction has existed for twenty-four hours (see literature of No. 18).

As a rule, gangrene begins in small insular areas at the convex portion of the herniated gut, i. e., that farthest away from the blood supply, and, finally, these coalesce. The constricted portion of the gut is usually the most severely damaged (*constriction furrow*). It is desirable to note that the disturbances of nutrition are not limited to the herniated portion of the gut, but that the oral gut is also similarly affected, especially if it is much distended (distention ulceration of Kocher[21]).

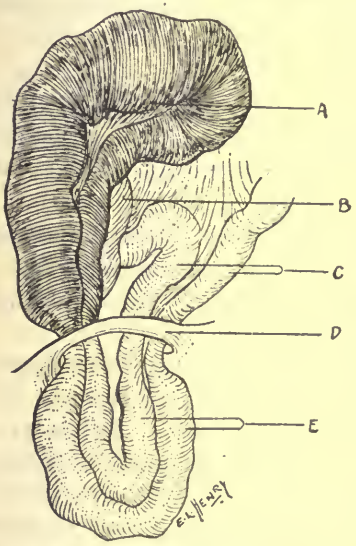

FIG. 919.— RETROGRADE INTESTINAL STRANGULATION (v. Witinghausen-Léjars).

A, Central portion of loop badly damaged by strangulation. *B*, Cecum. *C*, The two ends of the strangulated loop. *D*, Hernial port. *E*, Undamaged portion of gut lying in the hernial sac.

RETROGRADE CONSTRICTION. — The essential characteristic of retrograde intestinal constriction (*retrograde incarceration, incarceration sous annulaire, strangulation above the ring*) lies in the involvement of two portions of gut — one within the hernial sac, the other lying free within the abdominal cavity (Polya[23]).

In these cases the intra-abdominal sector may undergo more serious changes than the herniated. Some of them, no doubt, belong to the domain of "*reduction in mass*" or are due to a volvulus of the intra-abdominal loop of gut. As a rule, the strangulation is due to constriction of a portion of the mesentery which obstructs the circulation, especially when the herniated and the intra-abdominal loops possess a mesentery in common. Usually gangrene supervenes in a few hours.

The *clinical picture* of intestinal strangulation is exceedingly varied. As a rule, the patient ascribes the condition to an accidental cause, such as a severe exertion, an unusual muscular movement, or an overindulgence in eating.

Acute constriction is usually attended with a sudden increase in the size of the hernia and *severe pain;* the latter may be diffuse but is

most often localized at the site of the protrusion. In almost all instances, the lower bowel is emptied, but this does not relieve the feeling of distention the patient experiences; he is often conscious of moving wind in the abdomen which does not move beyond a certain place.

Nausea and vomiting occur at once and are not relieved by emptying the stomach. Within a few hours the hernial tumor becomes tense, hard, and tender; peristalsis is exaggerated and colicky pain and rumbling sounds develop; the vomitus becomes more abundant and soon is mixed with bile. At this time, the eructed gas is offensive in odor, and, finally, the vomitus is fecal in character, while the lower bowel ejects a series of foul stools.

The *general condition* of the patient is often surprisingly little affected; however, this is soon displaced by the typical picture of intestinal obstruction. In this stage of the affection patients often die in collapse.

In other cases the picture of *peritonitis* rapidly develops. This may occur early in the course of the strangulation, as the outcome of transmigration of bacteria through the damaged intestinal wall, or it may be the outcome of perforation of the gangrenous gut.

In the meantime, the *hernial contents* undergoes progressive changes. The protrusion becomes harder, red in color, and more tender; the inflammatory edema extends to the surrounding tissues. Finally, a restricted area bulges beyond the niveau of the surface of the tumor; fluctuation makes its appearance; the skin becomes discolored and ruptures, discharging putrefactive pus and foul smelling feces in large quantities.

The *rupture* may occur without involvement of the general peritoneal cavity, the latter being excluded by adhesive inflammation between the sac and its contents; in short, a localized putrefactive phlegmon is established. This event is immediately followed by relief of the symptoms, but, unfortunately, is rare. As a rule, the infective process extensively undermines the surrounding tissues and destroys them. Even this relatively gratifying outcome does not often occur; as a matter of fact, 95 per cent of the strangulated hernias, left to themselves, die.

The causes of death are:

1. *Shock,* due to the primary strangulation.
2. Toxemia occurring at the height of the strangulation (*stercoremia*).
3. Peritonitis.

4. Fecal phlegmon.
5. Remote complications, such as pneumonitis, etc.

The *symptoms of strangulated hernia* which have already been discussed in a general way may be arranged in the following groups:

1. *Changes in the hernia itself.*— A strangulated hernia is irreducible, enlarges and becomes hard, painful, and tender. If the hernia was originally irreducible, it no longer gives an impulse on coughing, and forcible expiratory compression does not influence its outline. Later, the picture of fecal phlegmon may develop.

2. The *disturbances due to obstruction.*— In completely constricted hernia, the bowels do not move and no gas is passed by rectum. At times, the lower bowel may expel a small quantity of feces, but this does not afford relief. Occasionally, when a high sector of gut is constricted, increased transudate is responsible for profuse fluid diarrhea (*cholera herniaire* of Malgaigne). When only the wall of the gut is constricted, the fecal current may not be interrupted.

In most cases, *meteorism* develops in the gut above the constriction. However, meteorism occurs only when the absorption of the gases from the oral gut does not keep pace with their formation. Many cases of complete intestinal occlusion do not exhibit meteorism; it is, however, constantly present when the circulation of the gut is interfered with. In the herniated gut meteorism is not manifest because of the increased tension in the sac; when the sac is opened the gut immediately "balloons" (local meteorism of Wahl). So-called *stasis meteorism*, which also develops in the herniated gut, is due to paralysis of the intestinal musculature.

The *vomiting* occurring in connection with strangulated hernia is similar to that of intestinal obstruction (see ileus) and is subject to the same laws. A strangulated hernia should never be permitted to develop fecal vomiting.

Decomposition of the intestinal contents and its absorption is an important factor in the affection; it leads to *stercoremia* and death.

3. The *influence of intestinal constriction and peritoneal irritation on the nervous system.*— The more acute the constriction the more marked are the symptoms ascribable to *trauma to the gut;* these are analogous to those attendant upon a severe blow in the abdominal region, and may be summarized as those of shock (*constriction shock*), together with *reflex vomiting* and *pain*. The last two symptoms may be absent when the colon and only the wall of the small gut are involved.

4. The *complications.*— To the symptoms of strangulation may be added those of *peritonitis* (local and general), and of fecal phlegmon, already discussed. *Pneumonitis* is usually lobular and is ascribed to embolism. *Anuria* is due to anhydremia.

The Diagnosis of Strangulated Hernia.— When a previously reducible hernia suddenly becomes irreducible, and this is attended with the symptoms stated above, the diagnosis is easy; in the absence of one or more important symptoms, the problem may become a difficult one. The best preventative against error is a systematic examination. One must not forget that more than one hernia may be present at the same time; there are a number of cases recorded in the literature in which the operative relief of constricted hernia was followed by the picture of intestinal obstruction and death, and autopsy revealed the presence of a strangulation remote from the field of operation. In doubtful cases, all the locations where hernia occurs should be examined.

The inability to reduce the hernia is the most important factor. Beyond this, the fact that neither expiratory abdominal pressure nor cough influence the protrusion is exceedingly important. In deeply located hernias, which are not susceptible to palpation, the sudden onset of pain and the presence of tenderness are weighty indications. Physical signs are unreliable in a general sense, but are valuable when positive emphysematous crackling upon palpation indicates perforation of the gut.

The *general symptoms* are of great diagnostic significance, i. e., the initial vomiting and shock may dominate the local disturbances. The latter is especially marked when the constriction is very firm, and, as already stated, in this event the local symptoms are likely to be unobtrusive. Strangulation of a small sector of the omentum is often attended with grave constitutional disturbances. However, in these instances the firm outline of the tumor gives a clue to its character.

Great difficulties arise when an *acute abdominal lesion* (localized peritonitis, torsion of the pedicle of a tumor, pancreatitis, etc.) develops in a person afflicted with an irreducible hernia. Differentiation in this regard is not always possible, and the surgeon must *perforce* act on the assumption that relief of the hernial element is justified.

The *diagnosis of retrograde strangulation* is rarely made before the operation. The presence of the lesion may be suspected when pain and tenderness above the hernia are disproportionate to the symptoms provoked by the hernia itself. If the conditions are suspicious in this

connection, the hernial port must be opened sufficiently to clear up the situation in this regard. How important this is is indicated by the fact the mortality in gangrene of the communicating loop is 50 per cent (Graser[17]).

The difficulties arising in connection with *false strangulation* have been emphasized by Clairmont,[24] who describes the condition as presenting the clinical picture of strangulation, but ascribes the sudden irreducibility of the hernia to a coincident intra-abdominal lesion, such as volvulus, peritonitis, etc.

Strangulation of a Herniated Intestinal Wall (Partial Enterocele).— In this variety of hernia (Richter's), fully described by Fowler,[25] the sac is small and the port narrow and rigid. As a rule, the contents consist of only a portion of the intestinal wall, although occasionally a process of omentum is also prolapsed. The enlarged sector of gut wall bears some resemblance to a diverticulum and for many years was regarded as such. The mechanism of the constriction is, in every regard, similar to that described above.

The *diagnosis* is not always easy. Reliance in this connection must be placed on the clinical picture as a whole.

Strangulated Omentocele.— The omentum is often prolapsed with the gut, but may be extruded and strangulated alone (5 per cent). The changes, due to strangulation, are likely to be very gradual.

The *symptoms* differ from those of enterocele with regard to intensity. The initial pain is, however, likely to be severe, especially in the region of the epigastrium. Palpation reveals the presence of an irreducible, hard, nodular mass. Vomiting occurs, and constipation is the rule. However, gas is usually expelled. The general condition of the patient is not seriously affected. The absence of intestinal bacteria in the hernial transudate explains the rarity of putrefactive infection, even after prolonged strangulation (five to six days), although sloughing and subsequent purulent inflammation is not uncommon. In a certain number of cases, spontaneous recession of the process occurs and the patient is left with an irreducible omental hernia.

In irreducible hernia of the omentum, the latter occasionally becomes twisted (*intra-abdominal omental torsion*). This is not readily diagnosticated until the abdomen is opened for the relief of an abdominal inflammatory process attended with tumor formation.

Clinical Forms of Strangulated Hernia.— From the description offered above it is patent that the phenomena attendant upon strangu-

lation, the character and degree of the constricting agent, and the severity of the symptoms vary widely. While this makes it logical that there are many cases of constriction which represent transitional forms in this connection, it is nevertheless permissible to divide strangulated hernia into *acute, subacute, and chronic,* from a clinical standpoint. The division would seem to need no farther discussion than to call attention to the mechanism of constriction and the explanation already offered of the various symptoms.

In the *most acute forms* (which are usually ascribable to elastic constriction) the systemic symptoms dominate the picture in the form of shock, soon followed by cyanosis, anuria, feeble pulse, persistent vomiting; and, unless relief is promptly afforded, a fatal termination ensues.

The *chronic form* of strangulation is attended with the gradual development of the picture of intestinal obturation. The surgeon must bear in mind, however, that at any time this may be effaced and replaced by that of acute strangulation.

The Treatment of Strangulated Hernia.— *Strangulation of a hernia must be relieved at once.* Procrastination in this regard is unforgivable.

For the purpose there are only two possibilities, (1) reposition by *taxis;* (2) *herniotomy.*

Taxis.— Although few surgeons employ taxis for the purpose of reducing a strangulated hernia, the fact that it occasionally succeeds justifies a description of the method. The manipulation must be executed with *great gentleness.*

Taxis may accomplish the purpose:

1. In very large hernias with wide ports in which the danger of rapid gangrene is not great, and the conditions make employment of operative measures dangerous.
2. In the first two hours of the constriction when damage to the contents is improbable.
3. In patients whose physical condition makes the operation particularly hazardous.
4. When even the most crude apparatus necessary to the operation is not available.

Taxis is contra-indicated:

1. When the strangulation has persisted long enough to make it probable that the contents is damaged.
2. When inflammation has developed.

3. In acute strangulation with shock, etc.

In the employment of taxis only one attempt should be made, and this should not last more than *five minutes*.

When taxis is decided upon it must be systematically carried out as follows:

1. The bladder is emptied — if necessary by catheterization.
2. The stomach content is withdrawn by syphonage.
3. The colon is emptied by copious lavage.

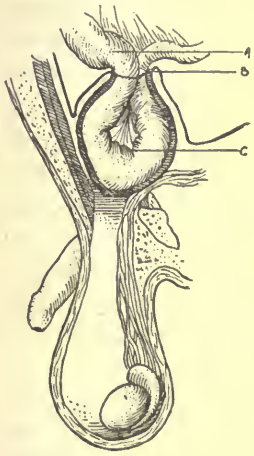

FIG. 920.— REPOSITION EN BLOC.

A, Oral end of strangulated loop. *B*, The reduced sac with its constricting neck. *D*, The strangulated loop of gut.

4. The patient is postured so that the hernial port is the highest point and the contiguous muscles are relaxed.

5. The patient is given a warm bath; this relaxes the muscles and cleanses the hernial area, which is desirable if a subsequent operation is necessary.

6. Complete narcosis is permissible but is not without its objections. It makes reposition easier and obviates pain, but is likely to invite dangerously forcible manipulations. It is probable that, when narcosis is employed, the added danger of the operative measures is not great, and preparation with this in view should be made.

The *technic of taxis*, as already stated, must be executed with the greatest gentleness. The neck of the hernia is grasped by one hand, the fundus with the other. Before attempting to reduce the protrusion an effort is made to deliver the point of constriction by gentle traction. After this, the reposition is attempted by alternate pressure on the neck of the sac and fundus. When the gut is reduced, the finger follows the course of the sac into its inner opening and the surgeon assures himself that the canal is empty. Recurrence is prevented by the immediate application of a suitable truss or a temporary pad and hip spica.

The Dangers of Taxis — False Reduction.— While the surgeon rarely uses taxis, he is often called upon to relieve its evil consequences. These consist in severe contusion with hemorrhage, rupture of the bowel, false reduction or reposition *en bloc*.

In false reduction the size of the hernia is lessened and it disap-

pears, but the constriction is not relieved. The following are the important forms of the condition:

1. Multisacular hernias in which the contents of the anterior loculus are forced into one more deeply located; this makes matters worse.

2. When the hernial contents, together with the sac and the constriction, is forced into the abdomen so that it either invaginates the parietal peritoneum or lies behind it.

3. The sac may tear at the site of the constriction and the gut be forced into the retroperitoneal space through this adventitious opening.

4. The constricting ring may be avulsed from the sac, remain clamped around the gut, and continue to strangle it.

When taxis succeeds in accomplishing the reduction, and this is not attended with rumbling of gas and gradual lessening in the dimensions of the tumor, but, on the contrary, the hernia suddenly collapses and this is accompanied by a tearing sound, suspicion of the occurrence of one of the conditions mentioned is justified. In these cases it is not infrequently possible to *palpate the uncorrected hernia* in the region of the port of exit in the form of a hard, tender mass. Relief is possible only by *herniocelio tomy*.

Herniotomy.— The operation is divided into the following steps: (1) The *skin incision*. (2) *Exposure of the sac*. (3) *Opening of the sac*. (4) *Abolition of the constriction and scrutiny of the contents*. (5) *Reposition of the contents*. (6) *Examination of the abdominal cavity* by passing the finger into it. (7) *Closure of the hernial port and canal*.

Applying this to an inguinal hernia, the following principles are to be observed:

1. The *skin incision* should permit of ample exposure of the hernial protrusion, including the hernial port. The *length of the incision* varies somewhat with the character and extent of the hernia and should expose the aponeurosis of the external oblique — an excellent guide to the subsequent steps of the operation.

2. *Exposure of the hernial sac* is best made by an incision at its neck, through known anatomical structures. The sac is readily identified by its fluid contents.

3. *Opening of the hernial sac* is an essential step in the operation and gives accurate information with regard to the condition of the contents. When the sac is exposed it is picked up with thumb forceps

and carefully incised. Mechanical cleansing of the contents is advantageous.

4. *Abolition of the constriction and scrutiny of the hernial contents* are very important measures in the procedure.

Abolition of the constriction "in the dark" (detridement) is no longer practiced. At this time all surgeons carefully divide the constricting tissues from without layer by layer "under the eye." By sectioning the arcade fibers of the external ring and the tunica vaginalis communis, the neck of the sac lies fully exposed and is now divided from below upward without making pressure upon the constricted viscus. Scissors, guided by the finger, are usually used for the purpose. This division must be sufficient to expose the "constriction furrow" and to permit of additional delivery of the endangered gut.

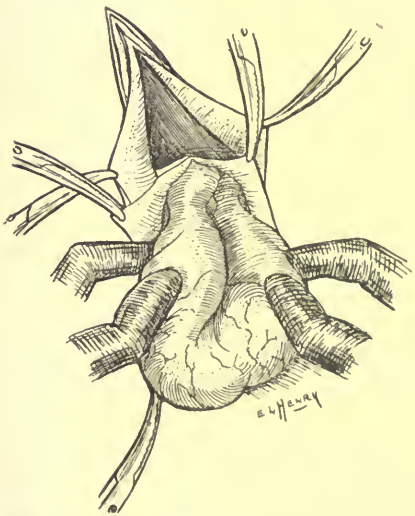

Fig. 921.— Scrutiny of a Delivered Strangulated Hernia.

The question whether the prolapsed gut should be returned is at times difficult to decide. In a general way, palpation gives more information than inspection. The living gut may contract and give the "feel of tension;" the dead is relaxed and feels like moist paper. Pulsation in the mesentery argues for viability. Color does not mean much — a bluish black gut may "come back." Dark gray spots mean gangrene. If there is doubt, a change in color may be awaited. Scarification with the needle is not permissible.

5. *Reposition of the contents* is executed with great care. Herniated omentum is amputated. Adhesions between the omentum and the gut are carefully separated. If reposition presents difficulties, it is best to deflate the loop of gut with the trocar; the opening is closed with a purse string suture. Reposition may be interfered with by twisting of the prolapsed loop on its axis, after the correction of which the return of the protrusion is readily accomplished.

6. *Examination of the abdominal cavity* by the finger, after reposition of the hernial contents, assures the surgeon that the central end of the canal is free and that the gut has not been reduced *en masse* or forced behind the peritoneum, nor through a split in the hernial sac.

7. *The procedure of today, when the gut is gangrenous,* consists in immediate resection of the necrotic viscus and entero-anastomosis by suture. For the purpose, the hernial port is enlarged, the gut is amply delivered, and the technic described in connection with entero-anastomosis (p. 1945) is carried out. The establishment of an artificial anus is only justified when the oral gut is in a condition of dynamic ileus. In these cases the radical operation will probably not be successful, because of the inevitable fertilization of the operative field, and, therefore, temporary tamponade of the superficial wound and subsequent closure by secondary suture is indicated. See statistics of Coley.[1]

8. Closure of the hernial port and suture of the wound is contra-indicated:

a. When the hernia is inflamed.

b. When enlargement of the hernial neck is attended with the discharge from the abdomen of a large quantity of turbid or foul smelling fluid.

c. When resection of the gut has been necessary.

d. When the field of operation has been contaminated with intestinal contents during the operation.

In all these instances the wound is temporarily tamponed with iodoform gauze and closed by suture a few days later.

While the skin may be closed in most instances, complete primary union in this class of cases is rare. For this reason a small silkworm gut drain should be left *in situ* for three days.

In the *after treatment* the use of opium is restricted to special indications, such as severe pain, restlessness, shock, etc.; its employment should be avoided, if possible.

A smooth convalescence obtains in about fifty per cent of the cases. If the gut has been much injured, it and the general system is still called upon to deal with the accumulation of toxic substances. This is combated by stimulation, hypodermoclysis and by the application of heat. The colon may be emptied by lavage. In a certain number of cases, *paralytic ileus* develops; this is met in the manner discussed under that head (p. 2018). The same may be said of a complicating local or general peritonitis.

In not a few cases the operation is followed by *gangrene of the testicle,* the outcome of injury to the spermatic artery. This demands removal of the organ, followed by drainage of the scrotum (Corner[26]).

The treatment of *spontaneous and artificial* fecal fistula has already been discussed (p. 1955).

Special Hernias — Inguinal Hernia.— The *anatomical conditions* predisposing to inguinal hernia are as follows:

1. Until the seventh month of fetal life a physiological hernial anlage exists in every male individual, i. e., the *processus vaginalis peritonei.* However, the gut rarely enters the process during intra-uterine life; as a rule, it is closed before the intra-abdominal pressure is sufficiently increased to force any of its contents into the process.

2. For some time after the testicle has descended the tissues concerned in obliteration of its course are likely to lack resistance to pressure and a portion of the gut readily finds its way into the process.

3. The fact that the spermatic cord passes through the inguinal canal is responsible for a certain weakness in this situation, which favors the escape of abdominal contents; this is contributed to by the funnel shaped depression located at the upper end of the obliterated canal (infundibular fascia).

4. The absence of musculature at the site of the inguinal canal renders it especially non-resistant; this is equally true in regard to the condition of affairs at the external ring, where hernia not infrequently occurs.

Inguinal hernias are divided into *indirect or oblique* and *direct or internal.*

Oblique Inguinal Hernia.— An oblique inguinal hernia emerges from the abdomen through the internal ring above and externally to the deep epigastric vessels; it then passes downward and forward (obliquely) parallel with Poupart's ligament, crossing the deep epigastric vessels, and finally extrudes through the external abdominal ring. Before the protrusion passes beyond the external ring, i. e., when it is *incomplete,* the term *bubonocele* is applied to it. A hernia which passes beyond the external ring and enters the scrotum is called a *complete* or *scrotal hernia,* in the male; in the female, the protrusion enters the labia majora and is called a *labial hernia.* Scrotal hernia is more frequently seen in the male than is the labial in the female.

In the normal inguinal canal the external ring barely admits the tip of the finger; in a large oblique hernia of long duration, the canal

becomes dilated and shortened, so that it may not always be readily differentiated from the direct variety.

The *anatomy of oblique inguinal hernia* may be described as follows: The protrusion leaves the abdominal cavity at the internal ring, which is situated about half way between the anterior superior spine of the ilium and the symphysis pubis, about one half inch internal to Poupart's ligament, and passes downward and inward to beyond the external ring. The sac rests upon a floor formed by the fascia trans-

Fig. 922.— Oblique Inguinal Hernia (Scrotal Type) (Johnson).

versalis and the peritoneum, between the layers of which lie the deep epigastric vessels. The roof of the canal is formed of the internal and external oblique muscles, the aponeurosis of the external oblique, the cremaster muscle, subcutaneous tissue and skin. The external ring is formed by the arched fibers of the aponeurosis of the external oblique, and varies in size in accord with that of the hernia.

As the sac passes downward, it always occupies a definite *relationship to the spermatic cord.*

An *oblique inguinal* hernia lies directly in front of and in intimate contact with the cord, the two being surrounded by a common layer of thin fascia (infundibuliform fascia). The cord consists of a number of vessels spread out on the posterior surface of the sac. In *direct hernia,* on the other hand, the cord is quite independent of the sac.

A scrotal hernia assumes a variety of shapes. A small one is usually cylindrical; the large ones are pear shaped; a very large hernia may obliterate the penis.

FIG. 923.— WOMAN AGED 40 YEARS, WITH RIGHT LABIAL HERNIA OF SEVENTEEN YEARS' DURATION (De Garmo).

A *congenital oblique inguinal hernia in the male* protrudes into a patent peritoneal vaginal process, so that the hernia and the testicle are enmantled by the same serous covering. As a matter of fact, only the sac is congenital.

In the *acquired form* the vaginal process is obliterated up to the internal ring; therefore, the hernia must push a new process of peritoneal sac ahead of it along the spermatic cord and does not lie in immediate contact with the testicle. The two are separated by two folds of peritoneum. This is schematically represented in Fig. 924.

As already stated, although the funicular process is often not oblit-

erated, congenital hernia is rare. There are, however, cases of genuine congenital hernia, and in these instances the contents is usually more or less adherent to the sac.

Although the vaginal process of the peritoneum descends with the testicle, it may also protrude into the scrotum when the testicle does not descend. In addition to this, the process may be only partially obliterated. It presents the following anatomical anomalies:

1. Obliteration only in the middle; the funnel remains open.
2. Obliteration at the spermatic cord; upper end open.
3. Obliteration only at its upper end.

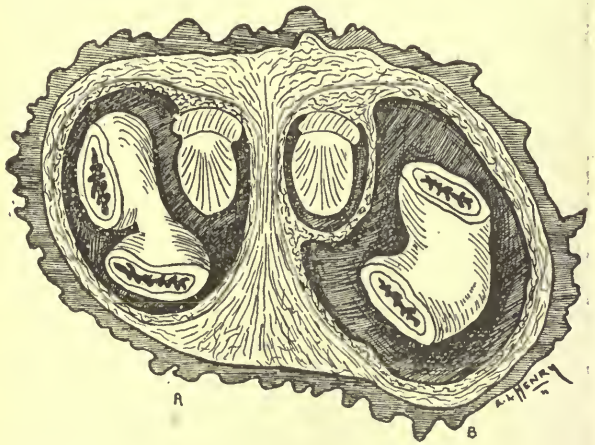

FIG. 924.—SEMISCHEMATIC SECTION THROUGH THE SCROTUM OF A MAN WITH TWO INGUINAL HERNIAS.

A, Congenital hernia; the testicle and gut lie in the same serous cavity. *B*, Acquired hernia; the testical lies in its own tunica vaginalis.

4. Almost total obliteration, with a small funnel provided with a valvular opening.

Atypical obliteration of the process is likely to be associated with undescended testicle. There is no doubt that most hernias are due to errors in obliteration of the vaginal process (Hansen[27]).

The simplest form of congenital inguinal hernia is that which prolapses into the bottom of the scrotum (*hernia vaginalis testicularis*). When the process is partly obliterated and the gut herniates into its upper end, the term *hernia vaginalis funicularis* is used.

Cryptorchism, hydrocele and hernia may be alluded to at this time. When the testicle cannot be identified the term cryptorchism is used.

When this happens the testicle may be arrested at any point in its course and a hernia occupy the vaginal process, which, in this class of cases, fills the entire scrotum.

When serous fluid collects in a patent vaginal process, the term *hydrocele vaginalis communicans* is employed. In this event, a portion of gut may prolapse into the fluid — *hydrocele hernialis.*

If the funicular process is closed above and fluid collects in the dependent tunica vaginalis, the ordinary *hydrocele testiculi* is produced. If the fluid also distends the upper portion of the process, the term *hydrocele testiculi et funiculi* is applied to it. If only that portion of the process in contact with the spermatic cord is distended with fluid (the process is closed at both ends), the term *hydrocele funiculi* (hydrocele of the cord) is used. It is easy to conceive that the obliteration may occur at several points in the course of the process, leaving "open spaces" between; if these fill with fluid, so-called, *hydrocele bi- or multilocularis* is established.

All of these conditions may be associated with hernial protrusions and must be borne in mind in connection with operative measures of relief.

The problem becomes especially complicated when an adventitious saculated projection of peritoneum, with its hernial contents, is invaginated into a tunica vaginalis which is the seat of a hydrocele of the tunica vaginalis or of the funicular process — *encysted hernia* (Wood[28]).

Direct Inguinal Hernia.— A direct inguinal hernia leaves the abdominal cavity at the inner inguinal groove, i. e., median to the deep epigastric vessels, and appears at the external abdominal ring. The sac is covered by the transversalis fascia, the superficial fascia, and the skin. The first may be separated by the protrusion and not form one of its coverings.

The hernia is usually round and, as a rule, even when very large, remains in the region of Poupart's ligament. This is of diagnostic value. At times, however, the hernia enters the scrotum, but is never intimately associated with the spermatic cord; the latter usually lies to the outer side of the sac. The hernia never enters the *tunica vaginalis communis spermatici et funiculi.* As this form of hernia usually develops gradually it is rarely strangulated.

Inguinal hernias in the female represent sixty per cent of the cases. In a general way, one may say that the vaginal process is smaller and is obliterated earlier in girls than in boys. The diverticulum of Nuck

has been found patent fifteen times in twenty-three operations performed by Berger.[29] Inguinal hernia occurs most often in women who have borne children — 772 cases out of 978 (Graser[18]).

As the hernia enlarges it creeps along the round ligament and enters the labium. Inguinal hernias in the female may be *incomplete* or *complete;* they are rarely direct (1 per cent), and at times are also associated with the various forms of hydrocele discussed in connection with hernia in the male.

The *treatment* is similar to that in the male. As there is no sper-

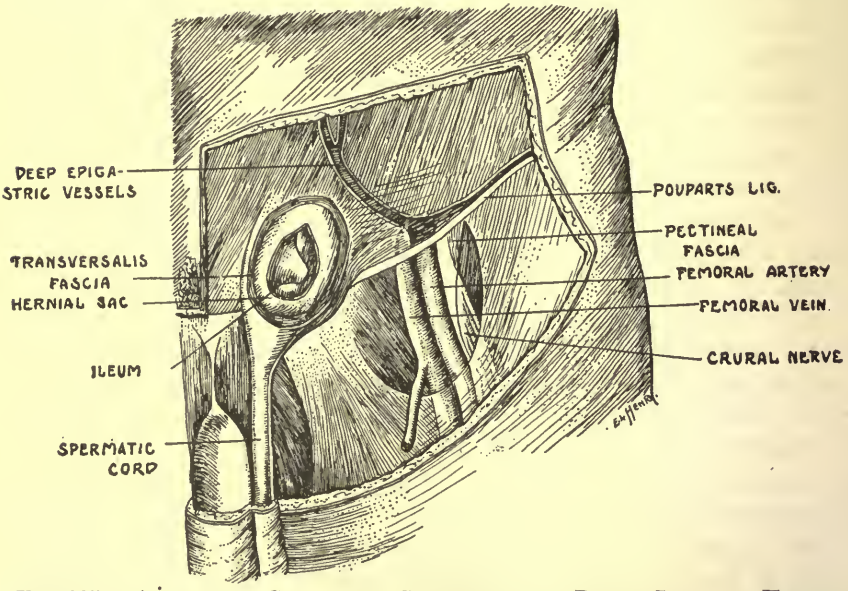

Fig. 925.— Anatomical Structures Concerned in a Direct Inguinal Hernia.

matic cord to deal with, the radical operation is very likely to accomplish the purpose.

Rare Forms of Inguinal Hernia.— Supervesical, intraparietal, and properitoneal hernias are uncommon forms.

Occasionally a hernia emerges through the groove between the urinary bladder and the lateral umbilical fold. The protrusion then descends obliquely outward in the abdominal wall, usually in the substance of the rectus muscle (Reich[30]). A number of peculiar hernias of this sort are described by Tilmann.[31] A discussion of the details in this connection cannot, for obvious reasons, be entered into here.

Intraparietal hernias (in a narrower sense) of the inguinal region consist in a protrusion with monolocular or bilocular sacs, which insinuate themselves between the layers of the abdominal muscles. This occurs most often in the space between the aponeurosis of the external and the internal oblique muscles. The pressure of the hernia often obliterates the fibers of the internal muscle (Grebel[32]).

Intraparietal hernias are found:

1. Between the external and internal oblique muscles.
2. Between the internal oblique and transversalis muscles.
3. Between the transversalis muscle and the fascia transversalis.

Properitoneal hernia has been clearly presented by Krönlein.[33] The sac is usually bilocular, the inner lying between the peritoneum and the transversalis fascia, and the outer in the inguinal or crural canal. In all cases there is a common orifice connecting both sacs with the abdominal cavity. Moynihan[34] is inclined to group the properitoneal with the interstitial or intraparietal forms; a viewpoint with which Coley[1] seems to agree. Most authors regard the condition as congenital in origin. The work of Cohn[35] argues strongly for the rationale of this conception.

The Treatment of Inguinal Hernias.— The so-called conservative treatment (trusses) has already been considered.

The indications for operative efforts at relief have also been taken up. This leaves for consideration the methods of operating.

The Radical Operation for Inguinal Hernia.— In a general way, one is justified in avoiding all operative methods in which the stump of the hernial sac or the plicated sac is fastened into the hernial port. In all instances, the sac should be removed as high as is possible. Simultaneous *castration* is rarely necessary. In sliding hernias it is, at times, permissible to reduce the testicle with the hernia, placing the former retroperitoneally and close to the canal (Graser[18]).

Until 1890, the methods of Czerny and Championnière have been largely employed. These, together with a number of others, possess only historical interest (see bibliography of No. 18). Here the description is confined to a description of the method of Bassini,[36] and the modification practiced by Bull and Coley.[37]

The *Bassini operation* is described by Coley[1] as follows:

"A straight oblique incision is made, beginning at the inner side of the anterior spine and one half inch internal to and parallel with Poupart's ligament, and ending over the center of the external ring. This incision is about three to three and one half inches long in adults,

and two and a half to three inches in children. The aponeurosis of the external oblique having been clearly exposed, a director is passed in at the external ring beneath the aponeurosis for a distance of about two and one half inches. The aponeurosis is then slit up with a scalpel upon a grooved director, great care being taken not to cut any underlying muscular fibers or the ilio-inguinal nerve. The aponeurosis is thereupon dissected back on the inner side, nearly to the edge of the rectus muscle, and on the outer side until the shelving process of Poupart's ligament is well exposed. The soft tissues on a level with the external ring, or slightly within it, are then grasped with a pair of thumb forceps, and the muscular fibers pushed aside by means of a pair of blunt pointed, curved scissors. The white tissues of the sac are thus quickly exposed and held by a pair of artery forceps. The sac and cord are then easily and quickly separated from the overlying tissues without hemorrhage, with the thumb and forefinger. In oblique hernia the sac will always be found anterior to the cord, to which it is closely attached by a thin layer of fascia (the infundibuliform fascia), which surrounds the two in common. By grasping the sac with a pair of artery clamps high up within the external ring, the cord is quickly and easily separated from the sac by means of the thumb and forefinger of the right hand, assisted by a small pad of sterile gauze. After one gets beneath the infundibuliform fascia the dissection is extremely easy, and the cord now lies well below and free from danger. The outer portion or roof of the sac is now cut across to see that it is empty; then the floor is cut and the upper end of the

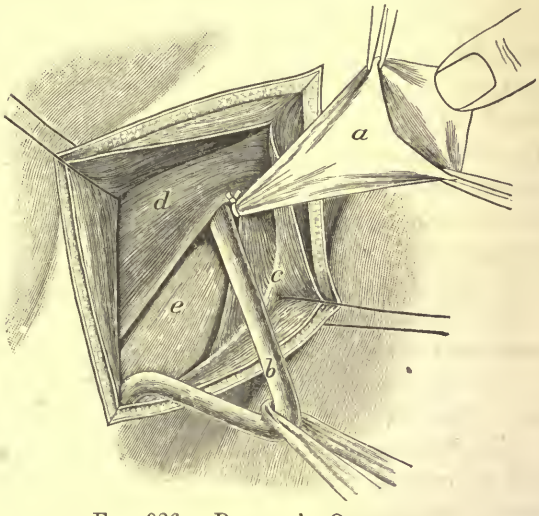

FIG. 926.— BASSINI'S OPERATION.

a, Sac dissected from cord, opened, examined, and neck ligatured; *b*, cord; *c*, Poupart's ligament; *d*, arched fibers of internal oblique; *e*, transversalis fascia.

sac is dissected well beyond the neck, or to a point where the sac widens out into the general peritoneal cavity. At this point the sac is transfixed with a catgut ligature and tied off (Fig. 926). If the sac be congenital, just enough of the lower portion to cover the testicle is allowed to remain, this being closed by continuous catgut suture. The internal oblique muscle should never be cut; in fact, no tissues are cut. Cutting the internal oblique muscle is entirely unnecessary for the complete removal of the sac. The cord is now held up out of the field of operation by means of a narrow strip of sterile tape or gauze. Next, the deep sutures of kangaroo tendon are introduced by means of a curved hagedorn needle. The first suture enters the internal oblique muscle nearly as far as the outer edge of the rectus muscle, and is so placed that it just touches the lower border of the cord when the latter is held vertically to the plane of the abdomen. The suture passes outward, first picking up the cremaster muscle, then the shelving edge of Poupart's ligament, and is finally tied. Three or four similar sutures complete the closure of the canal as far as the symphysis pubis (Fig. 927). I believe the lowermost suture is one of the most important in the series. My own practice is to insert the suture in such a way as to include the reflected portion of the external oblique aponeurosis as well as the conjoined tendon and the transversalis fascia. This apparently is not usually done, according to previously published cuts of Bassini's operation. In addition to the four or five sutures below the cord, I believe it a wise precaution to place one suture above the cord, passed through similar structures to those below. This is not strictly a part of Bassini's technic, but I believe it undoubtedly strengthens the canal in its weakest portion at the internal ring, thus lessening somewhat the liability to recurrence. The incision in the

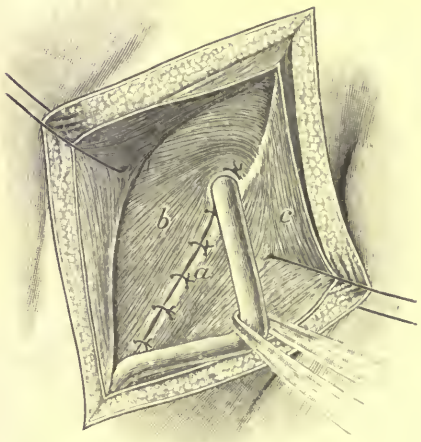

FIG. 927.—BASSINI'S OPERATION.

Arched muscular fibers and conjoined tendon (*b*) sutured to Poupart's ligament (*a*); *c*, aponeurosis of external oblique muscle.

aponeurosis is next closed by means of a continuous suture of kangaroo tendon of little finer caliber, the cord lying directly behind this layer. Care should be taken to leave sufficient room at the lower end or external ring to permit the cord to pass out without undue constriction. The skin wound is then closed without drainage, by means of interrupted sutures of fine catgut. These sutures are almost always found absorbed at the time of the first dressing at the end of one week. It should be stated that Bassini himself uses silk for the buried sutures."

The *Bull-Coley modification* of the Bassini method consists in passing the sutures in front of the cord, thus the latter is not transplanted (Fig. 928).

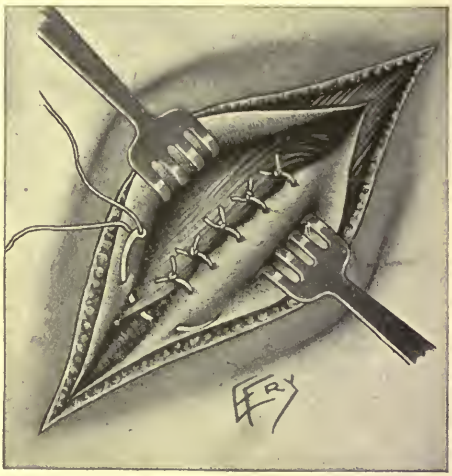

FIG. 928.— MODIFIED BASSINI'S OPERATION. Suture of internal oblique to Poupart's ligament without transplanting the cord (from Keen's Surgery).

In this connection Coley[1] says:

"A careful analysis of the cases thus far reported justifies the conclusion that the transplantation of the cord is not essential to the success of the operation, especially in children, while in adults, except in direct hernia, the results are nearly as good as in cases in which the cord is transplanted. The principal theoretic objection to the transplantation step of the typical Bassini operation, namely, the danger of atrophy of the testicle, resulting in sterility, has been shown to be without practical foundation. Atrophy of the testicle following Bassini's operation has never been observed at the Hospital for Ruptured and Crippled. Many of the children operated upon from ten to fifteen years ago have grown into adult life, have married, and are fathers of children.

"The time required for operation is about the same for the typical Bassini as for the modified operation, i. e., without the transplantation step. Either method can be performed in from eight to ten minutes by a surgeon familiar with the technic. The omission of the trans-

plantation step seems too slight a modification of the Bassini operation to justify anyone claiming it as an original method. It is the same method Bassini himself employed for the radical cure of inguinal hernia in the female. I still believe that the typical Bassini method will give slightly better results in oblique inguinal hernia in adults, and the transplantation step certainly has very great advantages in all cases of direct inguinal hernia."

Femoral Hernia.— In femoral or crural hernia the protrusion usually occurs at the inner side of the femoral vein; occasionally the hernia passes directly over the vessels (external femoral hernia of Narath[38]).

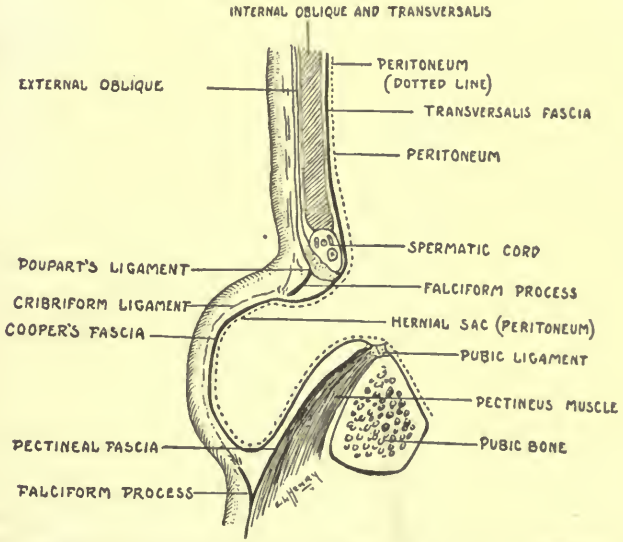

FIG. 929.— SCHEMATIC SAGITTAL SECTION THROUGH A TYPICAL FEMORAL HERNIA.

The work of Narath would indicate that this class of hernia is often due to trauma, and that it not infrequently occurs in connection with attempts at reduction of congenital dislocation of the hip.

The more important *anatomic relations* of femoral hernia may be briefly stated as follows: Poupart's ligament above, the pubic bone below (arcus femoralis), the pubic spine to the inner side. The former ends at the spine but is spread out in a fan shaped projection called Gimbernat's ligament. A schematic representation of the region is shown in Fig. 929.

In *acquired femoral hernia* the mechanism of the protrusion may be described as follows: The parietal peritoneum is gradually pushed

downward in the femoral canal, entering the sheath of the femoral vessels until it reaches the cribriform fascia, which it either penetrates or passes beneath. At the lower end of the femoral canal it curves forward, emerging at the saphenous opening to the inner side of the femoral vein.

There are, however, several atypical modes of exit; these may be grouped as follows:

1. *Hernia ligamenti Gimbernati* (Langier's hernia). The protrusion emerges through a gap in Gimbernat's ligament.

2. *Hernia femoralis pectinea* (Cloquet's hernia). The protrusion emerges through the internal ring and then makes its way through the loose pectineal fascia, and may separate the fiber of the muscle so that it lies as deeply as an obturator hernia (Dege[39]).

Fig. 930.— Femoral Hernia.

The patient was an old woman; the hernia was recurrent. No operation was done (Johnson).

3. *Hernia femoralis externa,* as already stated, protrudes to the outer side of the femoral vessels. Its peculiarity lies in the broad base it forms immediately below Poupart's ligament (Bähr[40]).

4. *Hernia femoralis retrovascularis.* The sac lies behind the femoral vessels.

5. *Hernia femoralis properitonealis.* The sac consists of two parts; one follows the usual course, the other, usually the larger, extends downward between the peritoneum and the pelvic fascia.

In most cases femoral hernia does not give rise to *symptoms* until the contents protrude from the lower opening of the canal. Very often women complain of vague digestive disturbances, which are

finally found to be caused by a small femoral hernia. The employment of a truss in femoral hernia is not satisfactory.

Strangulation occurs more often in connection with femoral than with inguinal hernia. As a rule, the neck of the sac is constricted at the edges of Gimbernat's ligament and the falciform process.

Gangrene of the gut occurs early. Taxis is rarely successful, and, in view of the early damage to the gut, is best omitted.

Herniotomy is best made at the inner border of the femoral vein. As the neck of the sac lies very close to the skin, the incision must be made with care. The gut must be very gently manipulated. When incising the neck of the sac, the knife is directed away from the vein (inward). In this way injury to the arterial ring (corona mortis) is avoided. Better still, division of the constriction may be executed by its free exposure and division "under the eye." It is occasionally necessary to divide Poupart's ligament; this freely exposes the internal ring.

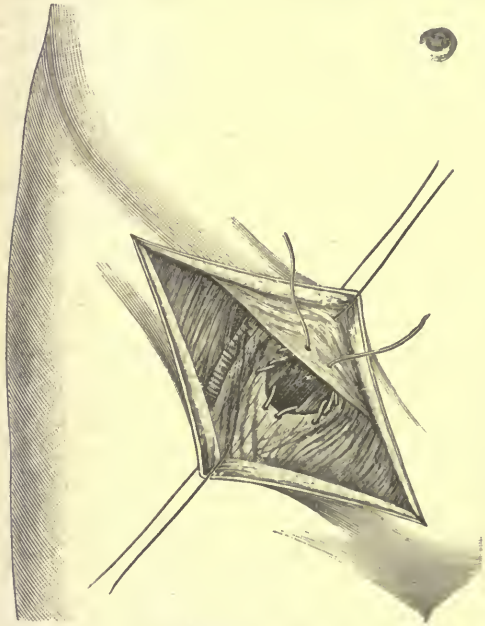

FIG. 931.—OBLITERATION OF THE FEMORAL OPENING BY PURSE STRING SUTURE.

THE RADICAL OPERATION. — At the present time the methods employed for the radical cure of femoral hernia may be divided into two main groups (1) those in which an effort is made to close the canal *by means of a suture* introduced in a variety of ways; (2) those in which the canal is closed by means of muscle, osteoplastic or heteroplastic methods.

The operations of the first group may be subdivided into those methods in which the canal is closed from the outer side, and those in which the canal is closed from within through an opening in the inguinal canal.

Coley[1] presents an instructive statistical review in this connection. Of the outer methods, that of Bassini[41] was practiced for a long time.

In this country, the method practiced by DeGarmo[42] is employed with considerable success. DeGarmo, after deligating the sac, uses a curved hagedorn needle threaded with kangaroo tendon of medium thickness; the suture is placed as follows: the needle is first passed through the inner portion of Poupart's ligament or roof of the canal, then downward, taking firm hold of the pectineal fascia and muscle, then outward through the fascia lata overlying the femoral vein, and finally upward, emerging through the roof of the canal about one quarter of an inch distant from the point of entrance (Fig. 931). On tying this suture, the floor of the canal is brought into apposition with the roof and the femoral opening is completely obliterated. Graser[18] uses a somewhat similar method. He, like most surgeons, regards the method as applicable only to small hernias, and has seen inguinal hernia develop after the operation.

Of the methods attacking the problem from the inguinal canal, the radical operation of Reich[43] is the least complicated and is widely employed. The method is really a combination and modification of those practiced by others, especially by Lotheisen and Föderl, Fabricius, Berard, etc. (see literature of Reich[43]).

The inguinal canal is opened in the same manner as in the Bassini operation (inguinal herniotomy). The transversalis fascia is opened; the sac is exposed, delivered and amputated as high as is possible. The spermatic cord is lifted and the arcade *fibers of the internal* oblique and transversalis muscles and Poupart's ligament are sutured to the periosteum of the pubic bone (Cooper's ligament). The method is particularly valuable in large hernias. For the plastic methods, which are rarely used at this time, see No. 44 of the bibliography.

Umbilical Hernia.— Hernia in connection with the navel has already been more or less discussed with congenital malformations of the abdominal wall, and will, therefore, receive only brief consideration at this time.

There are three varieties, (1) congenital umbilical hernia; (2) infantile umbilical hernia; (3) umbilical hernia of adult life.

Congenital umbilical hernia is really a failure of coalescence of the visceral abdominal plates during fetal life. Therefore, the coverings of the hernia consist of only a meager layer of Wharton's jelly, and a thin membrane or sac which is continuous with the parietal peritoneum.

The layers are exceedingly thin; the hernia very rare. Coley[1] says that of 75,000 hernias observed in the Hospital for Ruptured and

Crippled Children (New York), only four were of this nature. Ritterhous[45] has collected 153 cases.

The *operative treatment* is very simple. The edges of the abdominal wall at the base of the protrusion are refreshed (without opening the peritoneum) and sutured over the hernia.

Infantile umbilical hernia occurs in the first two years of life. After the cord is separated the residual opening is left large and the thin covering gradually permits of protrusion of the abdominal contents.

The *treatment* by pressure with suitable apparatus is very likely to be successful. Failing in this, the radical operation described in connection with the congenital type readily accomplishes the purpose.

Umbilical hernia of adult life is covered by peritoneum and connective tissue; subcutaneous fat is absent. The sac is usually very thin. The *contents* consist of omentum and ileum; occasionally the transverse colon and even the cecum are prolapsed. *Strangulation* is not rare.

The *treatment* with bandages or other apparatus is unsatisfactory and the maintenance of reposition is often impossible.

Of the radical operative measures, only those are worthy of consideration which permit complete visualizing of the contents and the definite separation of adhesions. A perceptible improvement in operative results followed excision of the umbilical port and suture of the refreshed muscles and fascia (omphalectomy of Bruns[46]). In large hernias, invagination of the hernial sac by layers of Lembert sutures, as worked out by Haynes,[47] gives excellent results. The method brings large surfaces of the coverings in apposition, and is especially useful in the relief of postoperative hernia following celiotomy.

The Mayos[48] make a large transverse elliptical incision and remove the redundant portion of the skin and fat, and fascia, together with the omentum. The fascial ring is then enlarged on either side until it reaches the edge of the rectus muscle. There the fascia, including the peritoneum, is overlapped by means of interrupted mattress sutures, so that the lower layer lies underneath and the upper layer externally. The edge of the upper flap is then fastened down upon the surface of the underlying flap by a continuous suture.

Graser[49] proceeds in a similar manner, but also mobilizes the recti muscles, which he sutures separately in a vertical direction. If this is feasible, it possesses a theoretical advantage over the Mayo procedure. Coley[1] regards the step as too complicated. Blake,[50] after exposing

and reducing the hernia, overlaps the fascia from side to side. In large ventral hernias the method possesses distinct advantages.

Obturator Hernia.—This form of hernia is uncommon. According to Coley,[1] none is recorded in the 75,000 cases seen at the Hospital for Ruptured and Crippled (New York). Berger[51] has collected 136 cases, of which 118 were in women.

The hernia emerges through the cleft in the upper portion of the obturator foramen, which gives egress to the obturator vessels and nerves. That is to say, the port of exit lies immediately beneath the edge of the pubic bone. As the hernia proceeds it is covered by the

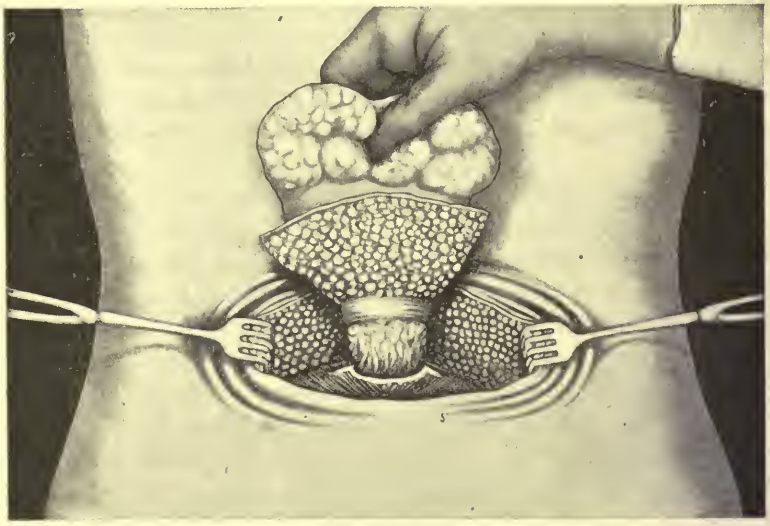

FIG. 932.—MAYO'S OPERATION FOR UMBILICAL HERNIA.

pectineus muscle, which, of course, makes its early recognition almost impossible. A tumor, if formed, corresponds to the site of femoral hernia.

The *diagnosis* is difficult. It is, however, less so in connection with strangulation. In the absence of a tumor, reliance must be placed on local tenderness, combined with the so-called Romberg symptom, which consists of pain, tenderness, and numbness along the course of the obturator nerve. The thigh is flexed and cannot be adducted. However, these symptoms are not constant and may be provoked by other conditions.

Strangulation of an obturator hernia presents the picture of internal

strangulation which, when associated with the local symptoms stated, may arouse suspicion of an obturator hernia.

The *treatment,* in view of the rarity of the diagnosis, is usually directed toward relief of the strangulation. In most instances, a median celiotomy undertaken for the relief of intestinal obstruction reveals the condition. It is usually feasible to deliver the gut from the abdominal cavity. Direct attaek upon the hernia is made through a large ineision along the inner border of the saphenous vein; the fibers of the pectineus are turned down from the pubic bone and the hernia exposed. Schwarzschild[52] closes the opening by turning an osteoplastic flap down from the pubie bone.

Ventral Hernia.— A hernia which protrudes through a portion of the abdominal wall where there is no preformed opening is called a ventral hernia. Hernias of this sort occur most frequently at the *linea alba* (above the umbilicus) and at the *linea semilunaris.*

Hernia at the linea alba is usually congenital in origin and consists in a diastasis of the reeti muscles. The condition is not infrequently found in multiparae.

The *congenital* form often heals spontaneously. In the others, partial closure takes place, leaving larger or smaller spaces, whieh give egress to collections of fatty tissue (*preperitoneal lipomata.*)

The clinieal interest in this class of cases lies in the fact that the condition is not infrequently attended with colieky pain, nausea, vomiting, and constipation. While, occasionally, a hernia of this sort contains colon and omentum, and the symptoms may be aseribed to fhis peculiarity, the disturbances are much more likely to be due to the presence of an intestinal diverticulum.

A careful examination often uneovers an irregularity in the course of the linea alba, i. e., a tumor the size of a hazel nut, whieh is tender and on slight pressure disappears into the abdominal cavity. Reduction reveals the presence of a small adventitious opening through whieh the tumor reappears when the pressure is released, especially when the patient coughs.

The *treatment* consists in exposure of the hernial site, excision of the fibrous tissue at the port and repair of the opening by suture.

Hernia at the linea semilunaris emerges where the aponeurosis of the transversalis becomes the posterior sheath of rectus abdominis, i. e., at the outer border of the latter muscle. At this site the vessels penetrate the abdominal wall, and it is here that the hernia occurs.

The protrusion presents the characteristics described in connection with hernias at the linea alba.

Ventral Hernia Following Celiotomy.— While this class of hernia is no doubt often ascribable to faulty technic in operative closure of the abdominal wound following celiotomy, it occurs most frequently in sequence to infection, and when drainage of the abdominal cavity has been necessary. Abel[53] found that if suppuration continued two weeks, forty per cent, if for three weeks, fifty-four per cent, and if for four weeks, eighty per cent of the cases developed hernia.

The *treatment* consists in the employment of the methods described in connection with umbilical hernia (p. 2079).

Lumbar Hernia.— A hernia protruding in the lateral portion of the abdomen between the crest of the ilium and costal arch, is regarded as a lumbar hernia. After a protracted discussion as to the port of exit, it would seem that Dowd[54] has shown that the hernia emerges through the triangle of Petit. This space is bounded below by the crest of the ilium, anteriorly by the external oblique, and posteriorly by the outer edge of the latissimus dorsi muscles.

The *diagnosis* is not difficult. The protrusion possesses all the characteristics of hernia discussed in the general considerations.

The *treatment*, other than the use of a truss, consists in exposure of the ring and its closure by means of a flap fashioned from the aponeurosis of the latissimus dorsi muscle. Coley[1] presents a series of instructive illustrations in this connection.

Ischiadic Hernia.—The major ischiadic foramen is filled in its greater part by the pyriformis muscle. The superior gluteal artery and nerve leave the foramen above the pyriformis muscle, while the inferior gluteal and the pudendal arteries and the sciatic nerve emerge below it; the minor ischiadic foramen gives exit to the obturator internus muscle, which is crossed by the pudendal artery as it reënters the pelvis.

Thus there are three places at which hernia may emerge, at the upper edge of the pyriformis, at its lower edge, and through the minor ischiadic foramen. All of the forms are very rare.

As a rule, the hernia protrudes through the upper opening and lies beneath the gluteal muscles, and occasionally presents itself at the lower edge of the larger muscle.

The *diagnosis* is not often made, unless strangulation occurs. In any event, the operative treatment consists in free exposure of the sac, its

ablation, and closure of the port of exit by suture, avoiding the contiguous vessels and nerves.

Hernias of the Pelvic Floor.— All hernias of the pelvic floor are ascribable to embryonic anomalies of the fold of Douglas, which consist of diverticula of the peritoneum; these are later occupied by intestine. The sac is covered by peritoneum and the pelvic fascia, and as it continues to progress, pushes downward the levator ani muscle or protrudes through it. In some instances, a hernia finds its way between the levator ani and the coccygeal muscles.

In this way the protrusion sinks into the side of the pelvis and presents itself at the ischiorectal fossa, either in front of or behind the transversus perinei muscle.

In the male, the hernia leaves the abdomen through the rectovesical space; in the female, it finds its way either in front of or behind the broad ligament. The anatomical relationship of hernias of this sort has been studied by Berger.[51]

The *diagnosis* in complete hernia is not difficult. The sac almost always contains gut and possesses the characteristics of this class of lesion discussed in connection with the general considerations (p. 2046).

The *treatment* is not satisfactory. An effort may be made to reduce the size of the pouch of Douglas through a celiotomy opening. Thomas[55] obtained relief by invaginating the sac and suturing it to the abdominal wall.

Diaphragmatic hernia is taken up in connection with Surgery of the Diaphragm (p. 1857).

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CHAPTER XIII

SURGERY OF THE LIVER

Anomalies of Position and Form of the Liver.— Transposition of the liver occurs as part of general visceral transposition (*situs viscerum inversus*). In cases of this sort the spleen is on the right side, and the thoracic organs, too, are usually transposed. However, the latter may not be the case. The malposition of the organ is usually recognized in the course of an examination made for other reasons.

As the condition is not of itself pathological, it is not attended with disturbances. Its chief practical significance lies in the confusing clinical picture presented in connection with diseases of the biliary passages and their complications.

Hepatoptosis.— Movable liver (hepatoptosis, constriction lobule, wandering liver) may be partial or complete (Ssaweljew[1]).

Partial hepatoptosis (constriction lobule) is ascribed to various causes (trauma, pregnancy, cholelithiasis, corsets, etc.). It may be so extreme that the constricted sector is attached to the liver itself only by a narrow isthmus consisting of fibrous tissue. The right lobe is more often affected than the left. Occasionally the isolated sector is the seat of congestion, which, in turn, becomes responsible for peritoneal irritation and its clinical picture, i. e., pain, tenderness, vomiting, and, occasionally, jaundice. The last is due to obstruction of bile ducts.

The *diagnosis* must be based on the manifest connection of the constriction with the liver. It will probably not be definitely established until exposed by celiotomy.

The *treatment*, in cases associated with a moderate degree of disturbance, consists in fixation of the lobule (partial hepatopexy) to the anterior abdominal wall by suture. In severe cases and in those in which the lobule has undergone pathological changes, excision of the mass is permissible (v. Haeker[2]).

Total hepatoptosis may be classed as follows: cases in which the liver is entirely separated from the diaphragm and may be replaced in its normal position by manipulation (wandering liver); and cases

in which the liver is displaced and becomes fixed in its new (abnormal) environment (fixed dislocated liver). The latter is rare and is found in women who lace their corsets tightly.

Wandering liver is usually associated with visceroptosis (Glenard's disease), in which the kidneys are especially movable.

The *symptoms* of hepatoptosis (often increased by jumping, riding, etc.) consist in pain in the epigastrium radiating toward the right shoulder. This is frequently relieved when the patient assumes the recumbent position. In severe cases pressure and dragging upon the abdominal contents are responsible for jaundice, ascites and digestive disturbances.

The *diagnosis* is based on the physical examination. The Röntgenographic findings in connection with the oxygen injection method are likely to be determining.

The *treatment* of movable liver, when associated with general visceroptosis, is nonoperative and consists in the measure discussed in connection with movable kidney (Part XIX, chap. i). Fixation of the liver by suturing its lower border to the costal margin, and scarification of the dome of the organ have been practiced with questionable success. Simultaneous plastic repair of the relaxed abdominal wall seems to be justified in selected cases (Depage and Meyer[3]).

Changes in the position of the liver from other causes may be arranged as follows:

Downward displacement may be the result of thoracic disease (pleurisy with effusion, emphysema, lung abscess, tumor of the mediastinum or lungs) or spinal deformity; it may also occur in connection with subdiaphragmatic abscess. *Upward displacement* is associated with ascites, abdominal tumors, and diaphragmatic hernia. *Posterior rotation* of the liver usually occurs with the upward, and *anterior rotation* with the downward displacement. The occurrence of either depends, of course, upon the presence or absence of perihepatic adhesions. This class of displacement of the liver, of itself is not important. The symptoms are those of the underlying causative factor. Clinically, recognition of the displacement is of value in the diagnosis of the cause.

Injuries of the Liver.— Experimental observations with regard to the processes concerned in the repair of wounds of the liver are recorded by Ponfick[4].

Injuries of the liver may be *classified* as open and subcutaneous.

From a *pathologico-anatomical* standpoint, three forms of *subcu-*

taneous injuries come under consideration; (1) *genuine rupture* of more or less of the liver substance attended with laceration of the capsule; (2) *décollement, or avulsion, of the capsule* with the formation of subcapsular hematomata; (3) so-called *hepatic apoplexy*, which consists in a number of disseminated or coalescing hemorrhagic foci which ultimately form cysts or abscesses.

Superficial injuries of the liver are finally followed by the formation of adhesions of the organ to contiguous parts.

Contusion of the right lobe occurs six times as often as that of the left. The convex surface of the organ is much more frequently involved than the concave. As is to be expected, the extent of the contusions or lacerations varies widely. Complete avulsion of a greater or lesser sector of liver tissue, the outcome of crushing force, has been repeatedly observed. Necrosis is usually the ultimate fate of the avulsed sector of liver tissue (Graser[5]).

Complete maceration of a portion of the liver presents a serious condition of affairs and, as the provocative force is of necessity severe, it is usually complicated by greater or less injury to other organs and parts (fractured ribs, lacerated lung, fracture of the skull, etc.). Occasionally this class of liver injury is attended with so-called liver cell embolism (Sehmorl[6]), which is a condition similar to fat embolism occuring in connection with fractures.

In cases of rupture of the liver with laceration of the capsule, the immediate, fatal result is due to internal bleeding. The fact that the veins of the liver are thin and are held open by their attachment to the surrounding cellular elements is responsible for the severity of the bleeding in this class of cases. In addition to this, neither the portal nor the hepatic veins have valves, and the hepatic veins communicate directly with the vena cava. The effused blood usually finds its way into the general peritoneal cavity, but this depends upon the location of the injury.

The *prognosis* in this class of cases is very grave. According to Edler,[7] death from hemorrhage occurs in the first twenty-four hours. Eighty per cent will die, if not relieved by operation (DaCosta[8]). The more remote dangers are those coincident with infection, with abscess formation, and with either local or general peritonitis. The infection may enter through an open wound or, in subcutaneous injury, may be hematogenous in origin. In rupture of the gallbladder or bile passages harboring pyogenic bacteria infection is almost certain to follow.

The *diagnosis* of rupture of the liver is based on the symptoms of shock, hemorrhage, and, if pleura, lung or diaphragm is involved, of respiratory embarrassment. Hepatic tenderness may be found in the subcutaneous cases, and the abdomen, though flat at first, later becomes rigid and distended. Jaundice has occurred in a few cases from extravasation of bile into the peritoneal cavity. If the patient is conscious, he may complain of pain in the region of the liver and possibly in the back. Dullness may be elicited in the right flank when the bleeding is intraperitoneal.

Regarding *operative measures of relief* in cases attended with bleeding, Deaver[9] states that "of the solid viscera of the abdomen the liver is most frequently injured and at the same time, the viscus most often recovers without operation."

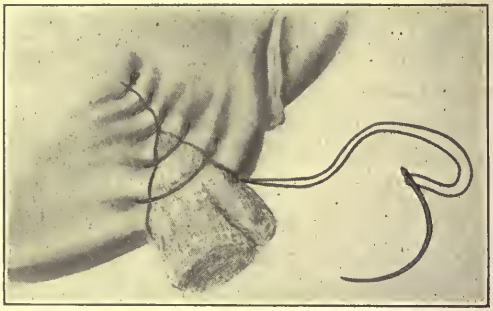

FIG. 933.—METHOD OF CHECKING HEMORRHAGE FROM THE LIVER BY COMBINED CATGUT SUTURING AND TAMPONADE.

In subcutaneous injury to the liver, the operation may be postponed but the patient must be kept under close observation. If the liver tenderness continues, and if the signs of the accumulation of blood in the right flank, together with increase instead of abatement of the signs of hemorrhage be presented, immediate operative relief should be resorted to. On the other hand, in the event of a sizeable open wound, or of obvious signs of crushing injury to the chest in the hepatic region, instant operation and arrest of the bleeding is indicated.

After the liver is exposed, either by the abdominal or the thoracic route (depending upon the nature of the injury), repair of the injury by mattress suture with heavy catgut, a large blunt needle being used for the purpose, is the procedure of choice. In severe cases iodoform gauze compresses may be sutured into the defect with catgut mattress sutures. The tampon is removed on the sixth or seventh day. The cautery does not accomplish the purpose.

Stab wounds of the liver are accompanied by violent hemorrhage. The possibilities in this connection are indicated by the degree of

bleeding attendant upon efforts at suturing this organ. Stab wounds do not present an especial clinical picture; occasionally, when the biliary passages are involved, bile escapes from the superficial wound. This class of injury is likely to be *complicated* by hemothorax or pneumothorax, and by injuries to the lung, stomach, intestines and gallbladder.

The question of *operative relief in stab wounds* of the liver is decided by the size and location of the wound, by the likelihood of injury to the bile passages (including the gallbladder), and by the presence or absence of injury of other viscera. Severe hemorrhage is always an indication for operation unless the shock is too severe. After the liver is exposed the bleeding is controlled as in contusion and rupture of the liver. To make the packing more effective, DaCosta[8] suggests first suturing the liver to the abdominal wall. A small wound of the gallbladder may be sutured; a large one demands cholecystectomy.

In *gunshot wounds* the liver may be lacerated, or penetrating, or the organ may be ruptured. The last type is most often fatal.

Soubeyran[10] reports the result of 26 cases of liver wounds. Twenty were immediately operated on with 11 recoveries and 9 deaths. In 6 cases not operated on, there were 3 recoveries and 3 deaths. Most of the injuries were shell wounds. The orifice was thoracic in 17 cases with 10 recoveries, and abdominal or lumbar in 9 cases with 4 recoveries. In 7 cases, the liver alone was injured. Shock was present in 7 cases. In 20 operated cases, the thoracic route was used in 7 (5 recoveries and 2 deaths); the abdominal route in 13 (6 recoveries and 7 deaths). Jaundice was seen in only 1 case, and the bile passages were found injured in 2. Death resulted in all 3 instances.

Soubeyran[10] gives as the two most important signs, in addition to the *symptoms* of internal bleeding, (1) *painful contraction* of the abdominal wall (*"défense musculaire"*) in front of the liver, with *hypersensitiveness* of the hepatic area; (2) marked pain on respiration.

Immediate operative efforts at relief are indicated when evidence of severe bleeding is presented. Abstention from interference is permissible when hemorrhage is slight. The Röntgenographic negative is of assistance in locating the projectile. Large wounds of the liver are sutured, a tampon of gauze being used to make pressure on the bleeding surfaces. Small perforations of the gallbladder may be sutured; large ones demand cholecystectomy. Drainage is provided for in all cases. Prolonged search for the projectile is inadvisable, on the ground that the hepatic tissue is very tolerant of foreign bodies and is capable

of protecting itself against sepsis. On the other hand, the liver is particularly susceptible to invasion by the gas bacillus (Soubeyran[10]).

The problem of "*débridement*" in these cases solves itself. If too much tissue is excised, the bleeding prevents further steps in this direction. Practically, the devitalized tissue is best removed by swabbing.

Gibbon[11] believes that "nature is more likely to stop the bleeding of a through and through gunshot wound of the liver than is the surgeon." This view the writer is not inclined to share. Anticipatory watchfulness is permissible only when bleeding is slight and when the nature of the wound makes infection highly improbable. In cases of severe bleeding speed in the operative procedure is of great importance.

Abscess of the Liver.— The *causation* of abscess of the liver is varied. The disease occurs more frequently in the tropics than in the temperate zone. In the latter it occurs most often in connection with *appendicitis* and *choleliathiasis*. Abscess of the liver is produced by microörganisms carried to it from the gut by way of the lymphatics or blood current. The excitants may also enter the organ through the biliary passages. So-called tropical abscess occurs most frequently in connection with *dysentery*. However, statistics in this regard vary widely (Kehr[12]). A certain number of abscesses follow trauma.

Of course, liver abscess develops in sequence to the acute infectious diseases by extension from the biliary passages; it may also be hematogenous in origin. The excitants of typhoid are particularly active in this regard (Sachs[13]). Direct invasion of the liver occurs in connection with gastric and duodenal ulcers and suppurative nephritis. Pyemic abscesses do not possess great surgical interest, while much importance may be attached to those secondary to ulcerative processes in the areas of origin of the portal circulation, i. e., perforating appendicitis, periproctitis, carcinoma of the gut, pancreatitis, etc. In this class of cases pylephlebitis thrombotica with secondary emboli of the intrahepatic portal veins is responsible for multiple abscesses in the liver. Occasionally a similar condition of affairs develops by way of the hepatic artery (gangrene of the lung, ulcerative endocarditis) and, in rare instances, pyogenic emboli gain access to the liver through the hepatic vein.

Abscesses of the liver are *multiple* or *single*. Amebic abscesses of long standing are likely to become sterile and are usually single. The non-tropical abscesses are usually due to the invasion of staphylococci

and the colon bacilli. The right lobe of the liver is most often invaded. The size of the abscess varies widely; occasionally the greater part of the liver tissue is destroyed. Metastatic abscesses and those developing from cholangitis are usually multiple. In a certain number of cases an abscess ruptures into a contiguous organ (lung, stomach, duodenum, kidney, etc.).

The *course* and *prognosis* of abscess of the liver varies with its cause. Tropical abscesses are likely to develop insidiously, and do not, as a rule, become manifest until months after the attack of dysentery has subsided. In not a few instances the presence of the abscess is not suspected until rupture into a contiguous organ occurs. This may be followed by recovery or, if the abscess ruptures into the pericardium, the vena cava, the peritoneum, etc., death rapidly ensues.

In multiple abscesses the prognosis is very unfavorable. In a few cases a number of the foci coalesce into a single, large abscess, in which event the outlook is less hopeless.

The *symptoms* of abscess of the liver remain latent in 33 per cent of the cases (Rouis[14]). Of Rouis' cases, the symptom complex (fever, pain, enlarged liver, tumor) was incomplete in 79 per cent. In only 8 per cent was it fully presented.

Fever is irregular in type; the temperature curve varies from the subnormal to several degrees above the normal and is often accompanied by chills and sweats (especially at night). However, fever and leukocytosis may be absent and, as a matter of fact, the latter is never marked except in cases of mixed infection. *Pain* is not severe and is usually dull in character. However, it becomes sharp when the process extends to the pleura or to the peritoneum. As the latter membrane is invaded, the upper, outer segment of the abdomen becomes rigid. In many cases the pain radiates toward the right scapula. Koch[15] calls attention to a gait peculiar to "liver cases." It looks as though the patient were carrying his abscess under his arm and feared to drop it. He proceeds with great care as though "feeling his way." One symptom of abscess, *enlargement of the liver, is never absent.* This varies, of course, with the location of the process and is readily discernible in the "oxygen Röntgenographic" picture.

The *diagnosis* of liver abscess may be exceedingly difficult, especially when there are multiple lesions and when a central abscess does not spread. Recognition is certain only when the process is superficial and fluctuation is ascertainable. *Diagnostic puncture,* while likely to be determining, is permissible only when the surgeon is pre-

pared to operate at once. Most surgeons regard exploratory celiotomy as less dangerous than aspiration. Hatch,[16] who has observed over 1,000 cases of abscess of the liver (in India), is very emphatic in denouncing aspiration as a dangerous maneuver. The physical examination must be thorough, especially with regard to the area occupied by the liver.

The *differential diagnosis* must be made to distinguish liver abscess from cholangitis, by the jaundice, in the absence of the history of gallstones; from malaria, by the atypical temperature curve and the examination of the blood; from multiple abscesses, by the history of any preceding disturbance (suppurative appendicitis, etc.), by the severity of the infection, the temperature curve and the leukocytosis; from lung abscess and empyema, by the mode of onset and by aspiration of the pleural cavity; from subphrenic abscess, by eliminating the possibility of the preceding focus, namely, appendicitis, rupture of a suppurative gallbladder, perinephritic abscess or perforated ulcer of the stomach or duodenum. When external rupture occurs, either through the skin, or by the air passages or bowel, examination of the character of the discharge determines the diagnosis.

The *treatment of localized abscess* is similar to that of abscess elsewhere. Drainage is secured by exposure of the liver by the abdominal or transthoracic route. The operation may be performed in two stages: (1) if no adhesions are present, the hepatic capsule is sutured to the peritoneum of the anterior abdominal wall, or, if the transthoracic route is followed, both layers of the pleura are sutured together, thus fixing the liver to the peritoneum of the diaphragm, and in twenty-four hours (after adhesions have formed) the abscess is incised; (2) if adhesions have already formed, immediate incision and drainage is practiced. If the abscess is not superficial enough to bulge and does not make its location evident, aspiration is permissible. The needle is left *in situ* and is used as a guide in dividing the hepatic tissue, the cautery being employed for the purpose.

The *treatment of multiple abscess,* because of the nature of the infection, is not likely to be satisfactory. However, occasionally a number of smaller, purulent foci lying contiguous to one another may be drained through a single opening by establishing communication between them by the finger (DaCosta[8]).

The *treatment of tropical abscesses* differs in no essential regard from that of a single abscess of pyogenic origin. Suturing the abscess to the peritoneum and secondary opening or immediate opening may

be practiced. The method of procedure is governed to a great extent by the location of the abscess. Occasionally it may be necessary to remove ribs or costal cartilages to provide the needed exposeure. The discharge from this class of abscess may not contain amebae at the time of operation, but it will the following day, due to the demand of the ameba for oxygen. Scrapings from the wall of the abscess always show active amebae (Fontain,[17] Wendel,[18] Smits[19]).

Cysts of the Liver.— Cysts of the liver are divided into *parasitic* and *non-parasitic* (true cysts). Of the parasitic cysts, the *echinococcus*

FIG. 934.— HYDATID CYST OF THE LIVER (From MacCallum's Text-Book of Pathology).

is the most common. The others (distoma hepaticum, lanceolatum, etc.) are not of surgical interest.

Hydatid cysts occur in the liver more frequently than in other portions of the body (65 per cent of the cases). They are due to the larvae of the tapeworm (tenia echinococcus). A description of the pathology of the parasite is admitted in the general part of this work (Part VII).

The *special pathology* of the hydatid cyst in the liver is presented by Ehrlich.[20] The cyst is usually single and is most often located in the right lobe of the liver. However, multiple cysts in this location

are not uncommon. Central cysts cause great deformation of the host; those originating near the periphery may become pedunculated, especially when they grow from the lower surface of the liver. In the latter instance they are readily mistaken for an enlarged gall-bladder. In most instances the cyst appears in the form of a protrusion, either in the right or in the left lobe of the liver, and the entire organ is greatly enlarged. The superimposed liver tissue is atrophied and the biliary passages are compressed. However, as the pressure on the ducts is very gradual, icterus in this class of cases is rare. Not a few of the cysts are ruptured by comparatively slight trauma.

Bacterial invasion of the cyst is not unusual. Its contents then rapidly undergoes fetid suppuration. Death of the cyst without infection may result in spontaneous shrinkage of the sac. In this event the contents undergoes calcification and is converted into an atheromatous paste. Occasionally the cyst presses on the portal vein and is responsible for ascites. It has caused obliteration of the vena cava. Cysts arising from the lower surface of the liver press on the stomach and duodenum. Perforation of the cyst may take place into the pleura and lung, and finally its contents may be expelled through a bronchus. This is followed by healing, unless the patient is strangled. In addition to this, the cyst may perforate into the portal vein, the inferior vena cava, the hepatic artery, the pulmonary artery, and may occasionally rupture into the biliary passages. When an infected cyst perforates into one of the blood passages stated, pyemia follows. Perforation into the bile ducts provides the picture of cholelithiasis; escape of the cyst contents through the biliary passages into the gut is followed by healing. *Per contra,* the entrance of bile into the sac kills the worm. If the bile is sterile, this is not attended with disturbances; if the bile is infected, an abscess is formed (Kehr[12]). The most favorable condition of affairs is produced when the cyst becomes adherent to one of the hollow viscera and ruptures into it. Perforation of the skin is a fortunate outcome but is not common. A cyst may rupture in more than one direction, for example, into the pleura and the stomach.

The *symptoms* of hydatid cyst of the liver, when the process is restricted in extent, may escape notice. Even large cysts do not cause disturbances until pressure is brought to bear on contiguous organs. A cyst of large dimensions causing considerable increase in the size of the liver may affect the general condition of the patient only slightly or not at all. The disturbances consist in a feeling of distention and

perhaps some dyspnea upon exertion. Real disturbances make their appearance when the cyst suppurates, a contingency attended with a picture very similar to that of abscess in this location. The pressure symptoms are, of course, dependent upon the compressed organ. Rupture into the pleura is announced by the clinical picture of acute pleuritis and, unless promptly relieved by thoracotomy, is very likely to be fatal. When the lung is adherent to the pleura and rupture occurs into the former, the symptoms of pneumonitis are presented. If this is followed by perforation into a bronchus, recovery is likely to occur. In this event the character of the process is usually not recognized until daughter cysts are expelled by coughing. Rupture of an infected cyst into the abdominal cavity is followed by the symptoms of peritonitis; rupture of an uninfected cyst provokes the picture of peritoneal irritation. However, the coincident absorption of toxins and ptomains produces evidences of systemic poisoning, in which urticaria plays an important rôle. Perforation into the biliary passages and consequent cholangitis are attended with icterus, especially when infection has been established.

In rupture into the urinary passages the daughter cysts appear in the urine and may be responsible for obstruction to the flow of the latter. Jönassen[21] reports a case of this sort, in which it became necessary to make an external urethrotomy.

The *diagnosis,* when the cyst is small, is impossible. The illuminating influence of eosinophilia does not eliminate the shadows of doubt. The serological examination is perhaps more determining. If the liver is the seat of a tumor, its smooth surface, elasticity, and more or less definite fluctuation should arouse the suspicion of hydatid cyst. The cyst is not tender unless it is infected. The tumor moves synchronously with respiration and is dull upon percussion. The much heralded hydatid fremitus is rarely appreciable. It was not discoverable in the 268 cases reported by Finsen.[22] At all events, it is also present in hydronephrosis, ascites, and in cystosarcoma of the liver (Kehr[12]).

Differentiation from true cysts would be established by diagnostic puncture, but, as has already been stated, the measure is not permissible. The dangers in this connection include toxemia from absorption of the echinococcus fluid (urticaria) and the implantation of the process into the general peritoneal cavity.

The diagnosis of suppuration in an hydatid cyst is easy when the presence of the cyst has been established before the infection and its attendant clinical picture develops.

Differentiation between an infected hydatid cyst and a *subphrenic abscess* is possible only by aspiration of the contents of the mass. In this event, the puncture may be made only when the surgeon is prepared to proceed with operative measures of relief at once.

Rupture of a cyst into the abdominal cavity is easily recognized when a tumor of the liver suddenly disappears and this is immediately followed by the evidence of peritoneal irritation and peritonitis. If this is attended with urticaria, the diagnosis becomes a certainty.

The *treatment* of hydatid cyst of the liver is operative. The older methods of injecting irritating fluids possess only historical interest (Langenbuch[23]).

Whether the operation is performed in one or two sittings, or whether the cyst wall is excised, depends upon the condition found when the lesion is exposed. Deeply located cysts which cannot be sutured to the abdominal wound, acutely infected cysts which have greatly reduced the resistance of the patient and demand immediate drainage, and multiple cysts should be subjected to the one stage operation. On the whole the surgeon will prefer the one sitting procedure. On the other hand, there is no doubt that the two stage operation is less dangerous. The most serious objection to it is that it makes simultaneous exploration of the abdomen difficult.

In both these methods, fixation of the hydatid cyst to the abdominal wall has its disadvantages, i. e., subsequent drag of the liver on the abdominal wall, the development of hernia, and the prolongation of the healing process. These disadvantages may be overcome by total excision of the echinococcus sac. However, this is not often feasible.

In the *two stage operation* the abdomen is opened by a vertical or transverse incision made over the dome of the protrusion. The surgeon then informs himself as to the size of the cyst, whether it is single or multiple, and as to the presence of adhesions. The portion of the sac to be opened is surrounded with gauze packing and the patient is confined to bed for ten days. At the end of this time the surface of the sac has become agglutinated to the wound and the cyst is opened with the scissors or the cautery and washed out with salt solution. The daughter cysts are thus readily evacuated; occasionally it is possible to deliver the mother cyst by gentle traction with forceps. Force (for fear of trauma to the liver tissue) must not be employed. A drainage tube is inserted into the cyst cavity, and, as time goes on, this is gradually shortened until healing is complete.

In the *one stage operation* the cyst is opened at once. The wall of

the cyst is either sutured to the wound of approach before the sac is opened or the latter step is accomplished first and the former follows. In either event, the tension of the cyst should be reduced by aspiration after it is exposed. Thus suture is more readily accomplished. Similar methods are employed when the cyst is infected, especial care being taken to obviate soiling of the peritoneum.

As healing by the drainage method is prolonged, an effort has been made to achieve *primary repair* (Bobrow[24]). The contents of the cyst is evacuated, the connective tissue capsule is obliterated by suture and either fixed in the celiotomy wound or the abdomen is closed over the repaired sac.

When the cyst is located at the surface of the liver or happens to be pedunculated, it, together with its connective tissue capsule, may be enucleated *in toto* (Körte,[25] Garré[26]).

Hydatids located at the convex border of the liver may be approached by the transpleural route (p. 1857). Israel[27] reports a case of this sort. Resection of the eighth to the eleventh costal cartilages also makes this area accessible.

Chorerrhagia follows echinococcectomy in more than one half of the cases, and is responsible for a fatal outcome in from 7 to 16 per cent. Spontaneous or surgical evacuation of the capsule opens the minute bile ducts, which makes possible the discharge of bile into the wound. It is probable that, when spontaneous healing does not occur, the hepatic or common duct is obstructed. Kehr[12] has caused biliary fistulae of this sort to heal by temporary drainage of the hepatic duct. In one case removal of a stone from the common duct was followed by closure of the hepatic fistula.

Multilocular cysts are susceptible to measures of relief only when the lesions are confined to a portion of the liver amenable to resection.

Non-parasitic Cysts of the Liver.— Compared with parasitic, true cysts of the liver are rare. Many are not discovered until autopsy is made. The small *mucous retention cysts* of congenital origin are not of surgical importance (v. Recklinghausen[28]). *Dermoid cysts* of the liver and lymph cysts are the other two kinds found in the liver. Leppmann[29] describes cysts which are the result of occlusion of the smaller or larger bile ducts. He groups these with the epithelial lined cysts which are generally regarded as *"multiple cystic degeneration"* of the liver. *Cystadenomata* develop from the glandular elements of dilated bile ducts. Hoffmann[30] has collected 18 cases of true cyst of the liver.

The *symptoms* of true cysts of the liver are analogous to the hydatid.

The *treatment* consists in the one stage operation described in connection with echinococcus. Contamination of the peritoneum is a factor only when the cyst is infected. This is likely to be the case when the lesion originates from the biliary ducts.

Chronic Hepatitis.— Chronic hepatitis, with atrophic cirrhosis, has been subjected to operative measures of relief; i. e., the Talma-Drummond operation (Bunge[31]). The maneuver is destined to unburden the portal circulation and relieve ascites. The technic of the operation is simple. The abdomen is opened in the median line and the omentum is sutured to the parietal peritoneum. Some surgeons endeavor to establish additional adherence of the visceral and parietal peritoneum by irritating the latter with gauze sponges. The operation has been followed by relief in a small percentage of cases of uncomplicated hepatic cirrhosis. In coexisting nephritis the measure is of no avail.

Tuberculosis of the Liver.— Tuberculosis of the liver is rarely recognized *in vivo*. It occurs most often in connection with general miliary tuberculosis. Infection by direct extension from the gut is not uncommon, and the organ may be affected as a part of tuberculous peritonitis originating in one of the other abdominal organs (MacCallum[32]).

Occasionally a so-called *solitary tuberculous abscess* is uncovered during exploration of the abdomen for the relief of an indefinite liver lesion. In this event, drainage of the abscess and the injection of iodoform emulsion may be practiced.

Actinomycosis of the Liver.— Actinomycosis of the liver is usually secondary to that of the intestinal canal. The pathological picture is that of a "honeycomb structure of connective tissue saturated with pus." It presents the *clinical picture* of hepatic abscess and, in the absence of a history of actinomycosis elsewhere in the body, can be *diagnosticated* only by recognition of the ray fungus in the exudate.

The *prognosis* is grave, but is dependent upon the extent of the disease and the seat of the underlying affection.

The *treatment* is similar to that of actinomycosis in general (p. 411). Resection of the liver has been practiced, but is a daring maneuver (Auvray and Terrier[33]).

Solid Tumors of the Liver.— *Lues of the liver* appears in two forms.

The *diffuse* form of the disease (nodular liver) is probably originally gummatous, the process gradually extending and infiltrating the liver substance. It has no surgical significance.

Gumma of the liver may be multiple or single. The lesion varies

in size from that of a pea to that of an apple. It appears most often at the site of the suspensory ligament and at the entrance of the portal vein. Coincident ascites is explained on the basis of general connective tissue hyperplasia or of the pressure made on the portal vein.

The *diagnosis* of syphiloma of the liver (Röntgenogram-Wassermann reaction) is not often made. The presence of a tumor in the liver may be recognized, but its character is likely to be uncertain. This, together with the fact that primary carcinoma of the liver, perhaps susceptible of resection, may develop in a luetic patient, justifies exploration. In addition to this, removal of an accessible gumma of the liver is a permissible maneuver (Haubold[34]).

The *benign tumors* of the liver are usually harmless and demand efforts at relief only when they are of such size as to cause pressure disturbances. Many of these have been successfully removed (Anschütz[35]). Adenomata, because of their tendency to become malignant, should be freely excised (Keen[36]).

Solitary adenomata of the liver are at times the seat of hemorrhages of considerable magnitude. These are followed by cyst formation. In other instances the growths become infected and present the picture of abscess of the liver. *Hypernephromata* develop from displaced remnants of the adrenal gland, and are likely to be very malignant.

Carcinoma of the liver is rarely primary. Of 713 cases studied by Orth[37] only 2 originated in the liver.

Primary carcinoma appears in the following forms: (1) *massive*, involving large portions, and well defined from the unaffected liver; (2) the *infiltrating*, consisting of a single lesion with secondary deposits in its vicinity; (3) the *nodular;* (4) the *cirrhotic.*

Primary carcinoma of the liver may *originate* in the bile duct epithelium or from the liver cells themselves.

Secondary carcinoma is the most common tumor found in the liver. It is always multiple and due to metastatic growths derived from carcinoma of the gastro-intestinal tract. Occasionally, by reason of the anastomosis between the portal and uterine or ovarian circulation, malignant involvement of the uterus and ovary may cause secondary deposits in the liver. The condition itself is inoperable, but it is of the greatest importance in deciding whether or not an operation upon the intestinal tract should be undertaken. For this reason, before extirpating any malignant abdominal tumor, it is a sovereign rule to examine the liver for metastases. If these exist, it would be futile to perform extirpation of the primary growth. The nodules vary in size and

shape. At first, if the process presents on the surface, the nodules are grayish white with convex or flattened surfaces; later, because of central necrosis due to blood impoverishment, they become yellow and umbilicated.

There may be no *symptoms* of the lesion until there is gross evidence (palpable tumor) that the liver is involved. Occasionally a slight, sudden pain in the hepatic region, accompanied by chill, temperature and, perhaps, jaundice, indicates the lodgment of one or more malignant emboli in the liver. Jaundice and ascites are more frequent in secondary cancer of the liver than in primary. In a certain number of instances a secondary cancerous deposit from carcinoma of the gall bladder may be adjacent to this organ and it may be possible to remove it. Where there is a linguiform lobulation (Riedel's lobe) it is at times feasible to remove the gallbladder together with the invaded portion of the liver *en masse*. However, recurrence is the rule.

Resection of the liver for carcinoma is an hazardous undertaking because of the bleeding. The usual methods of hemostasis and those taken up in connection with injury of the liver are likely to prove ineffective. Tumors of the left lobe of the liver may be removed by encircling the entire lobe with a rubber tube, as is done with the uterus in Cesarian section. In all cases tamponade becomes necessary. Efforts have been made to obtain a bloodless field by temporary ligature or compression of the hepatic and portal vessels as they enter the under surface of the liver. Daring as the maneuver seems to be, it has accomplished the purpose in not a few instances (Narath[38]).

Sarcoma appears in the liver in the form of a diffuse infiltrating massive tumor. It is usually metastatic from some other location. The growth is very rapid. It occasionally arises from the mesodermal connective tissue of the liver. The most frequent variety is *melanotic sarcoma* of the liver, which has its origin in the uveal tract of the eye or in pigmented moles of the skin that have undergone malignant change. Whether these tumors are of epithelial or mesodermal origin is uncertain. Many cases suspected of being primary sarcoma of the liver are of this variety, and the statistics are not to be depended upon because examination of the uveal tract has not been made. In the young, care should be taken to differentiate sarcoma from congenital luetic hepatic involvement.

The *treatment* can be only palliative.

Of the other tumors of the liver, *aneurism* of the arteries of this organ deserves mention. If, during exploration, a lesion of this sort

is uncovered, it may, according to Narath,[88] be subjected to treatment, with the following reservations:

1. The common hepatic artery may be deligated, provided one of the collateral vessels is patent.

2. Ligature of the hepatic artery central to the origin of the gastrica dextra is permissible only in emergency.

3. It is always preferable to tie either the right or left branch of the hepatic artery; ligature of the main trunk is likely to be followed by necrosis of the liver.

Surgery of the Biliary Passages — Special Anatomical Considerations. — A knowledge of the normal anatomy of the liver and biliary passages does not suffice the surgeon when he is confronted with deformations, the outcome of inflammation and adhesions. In addition to this, the biliary passages (especially the cystic and common ducts) and more particularly the hepatic and cystic arteries are frequently anomalous in their arrangement. All surgeons know that the hepaticoduodenal ligament contains the common bile duct, the hepatic artery, and the portal vein. However, in making a choledochotomy, one's knowledge of anatomy is not to be relied upon as regards the avoidance of injury to these structures. This can be obviated only by approaching the area under consideration through an ample incision, which permits of visualizing the various structures.

The gallbladder lies at the anterior edge of the right sagittal fossa between the quadrate and right lobes of the liver. An incision beginning at the edge of the costal arch, at the eighth to the ninth costal cartilage, and carried downward along the outer border of the right rectus muscle uncovers the gallbladder under normal conditions. As the surgeon deals with abnormal conditions, he often finds variations in the position of the organ. It may be entirely covered by the liver, located at the median line, and it occasionally (excluding cases of *situs transversus*) lies on the left side. It has been found entirely embedded in liver tissue. Absence of the gallbladder has been encountered only once by Kehr.[12] The organ is often located low in the abdomen (cecal region), and in not a few instances has been discovered in the lumbar region. These variations occur in connection with the changes in the volume and form of the liver (constriction liver, etc.); adhesions, too, are responsible for the malposition. The size of the gallbladder is exceedingly variable. In cases of stone in the cystic duct and in hydrops it may attain the dimensions of an infant's head, and may be mistaken for an ovarian tumor. On the other hand, the gallbladder

may, as the result of chronic inflammation, shrink to the size of a cherry. As to the structure of the gallbladder, the reader is reminded that its peritoneal covering does not enmantle the organ where it comes in contact with the liver. It is important to remember, especially in extirpating the gallbladder, that it is supplied by the cystic artery which divides into two branches at the neck of the organ; these branches follow a subperitoneal course to the fundus. In addition to this, a third artery (accessory cystic) arises either from the gastric or hepatic and enters the free surface of the gallbladder. In most instances the gallbladder is closely apposed to the under surface of the liver. Occasionally it possesses a mesentery, in which event, the organ is more or less movable (wandering gallbladder), and may become strangulated as the outcome of torsion. The gallbladder tapers to a duct — the cystic — which is about 3 cm. in length. Its lumen is thrown into folds (folds of Heister). This arrangement makes it difficult, if not impossible, to pass a sound through it. The cystic opens into the hepatic duct. A small gland situated at this point is often mistaken for a concretion. The anterior wall of the foramen epiploicum (foramen of Winslow), which is made up of the ligamentum hepatoduodenale, contains on the right the hepatic duct, next, and slightly behind, the portal vein, and to the left, the hepatic artery. The hepatic duct is formed by two (right and left) branches. However, several of these may converge into the main duct. The ductus choledochus is divided into a portion lying above the duodenum, about 3.5 mm. in length; a portio pancreatica (retroduodenal), about 2.5 mm. in length; and a portio duodenalis (intraduodenalis or terminalis), about 1.5 mm. in length. The upper section of the duct is the freest and is particularly accessible to invasion by the surgeon. However, due consideration must be shown the gastroduodenal artery, which crosses the duct in this location. The retroduodenal portion of the duct is best approached by the transduodenal route.

Normally, the bile duct is the size of a lead pencil and possesses thin walls. As the outcome of the presence of stones and infection, it becomes dilated and its walls become much thickened. The duct is best located by exposing the cystic duct and identifying the lateral edge of the duodenum. The forefinger of the left hand is then introduced into the foramen of Winslow (which unfortunately is often obliterated by adhesions), and by lifting the structures the various parts may be recognized. Aside from the one already mentioned, a

large, long gland lies between the bile duct and the duodenum and another is situated above the hepatic artery. These are often much enlarged. The relationship of the duct to the pancreas is taken up *in extenso* on page 2086.

The important consideration in connection with the papilla duodenalis relates to the frequent presence of a diverticulum central to it. If this is present, a stone lodged within it obstructs the flow of the bile and pancreatic juice, and in the latter event is likely to be responsible for pancreatitis. The work of Ruge[39] in this connection is exhaustive.

The gallbladder is not essential to life. If it is removed together with the cystic duct, it is not regenerated. When the cystic duct is left *in situ*, it dilates and forms a "sort of gallbladder."

Injuries of the Biliary Passages.— Experimental observations seem to show that the entrance of bile into the abdominal cavity is followed by stormy disturbances, but that fatal inflammation is not provoked. If, however, the effused bile is derived from infected biliary passages, peritonitis, the extent of which is decided by the character of previously established adhesion, occurs.

A dilated gallbladder may be ruptured by comparatively slight trauma.

The *symptoms* of traumatic rupture of the gallbladder do not differ widely from those presented in connection with injuries of the liver (p. 2086). Rapid emaciation it attributed to the loss of fluid. Icterus does not appear until the third or fourth day. The effused bile is absorbed by a healthy peritoneum. In the presence of chronic peritonitis the bile collects and icterus does not develop. As time goes on, the abdomen becomes distended. In penetrating wounds the discharge of bile argues for injury to the biliary passages rather than to the liver. Courvoisier[40] presents an extended study of the subject, together with a complete bibliography.

When the diagnosis of injury to the biliary passages is established, the abdomen is freely opened, an incision parallel to the costal arch being made for the purpose. Circumscribed openings in the gallbladder are closed by Lembert sutures. In severe injury to the gallbladder or cystic duct, cholecystectomy is permissible. An injured common duct is repaired as after choledochotomy. Drainage is provided for in all instances.

Of course, injuries of this class are very likely to be associated with

more or less trauma to the liver. This situation is met in the manner stated above (p. 2086).

Inflammation of the Biliary Passages and Gallstone Disease (Tuberculosis and Actinomycosis of the Biliary Passages and Idiopathic Cystic Bile Duct).— Inflammation of the biliary passages cannot readily be separated from gallstone disease (Cholelithiasis). It is true that gallstones may develop without infection (the radial cholesterin stone of Ashoff[41] in sterile bile). However, the gallstones that the physician, and more especially the surgeon, has to deal with are always associated with inflammation, *the outcome of infection.* Surgical efforts at relief are influenced by the degree and character of the infection, not by the fact that a stone is present.

Every inflammation of the biliary passages — cholangitis — is caused by infection which either ascends from the gut (bacterium coli) or descends from the blood (bacillus typhi). The preëxisting foreign body theory of Riedel has been abandoned. The presence of stones, ascarides, etc., favors the invasion of bacteria. Trauma is responsible for infection in the same way. All of the acute infectious diseases are likely to be followed by cholecystitis or cholangitis.

The *pathological anatomy* of all forms of inflammation of the biliary passages is similar to those occurring in the appendix and need not be repeated here. The various forms have been studied exhaustively by Ashoff and Bacfeister.[41]

Inflammations of the biliary passages heal in the same way. However, acute cholecystitis is much more likely to merge into the *chronic form.*

The *symptoms* of acute and chronic cholecystitis do not carry out an accord with the existing pathological process. Acute gangrenous cholecystitis may present the clinical picture of ileus or peritonitis, or may even, when the gallbladder is gangrenous, give rise to but slight disturbances. As a rule, however, the symptoms are the same as in appendicitis (fever, chill, abdominal rigidity, severe pain). Icterus is slight or absent. In most instances a pancreatic lymphadenitis is also present. Serous and seropurulent cholecystitis may follow as stormy a course as the gangrenous form and may, like the latter, give rise to only slight disturbances. Fever may be absent in suppurative cholecystitis.

Chronic cholecystitis is more difficult to recognize than the acute form. The gallbladder shrinks, its mucosa ulcerates, and even though

the organ contains pus, it is usually not palpable. Neither acute nor chronic cholecystitis is attended with enlargement of the liver.

Inflammation of the interior of the gallbladder frequently extends to its surroundings. The process migrates through the spaces of the ducts of Luschka and through minute perforations, and becomes responsible for pericholecystitis. This results in abscess formation or in the development of adhesions. The latter are often responsible for agglutination and deformations of the pylorus, duodenum, omentum, etc., which, in turn, give rise to distressing disturbances. At this time the reader is warned that simple separation of the adhesions does not afford relief. It is necessary to remove the gall-bladder or to make a gastro-enterostomy and a simultaneous pyloric exclusion. In a certain number of cases both are necessary.

Intraperitoneal rupture of the inflamed gallbladder is uncommon. The organ is usually encased in the omentum and the complicating peritonitis is localized.

Perforation of a suppurating gallbladder into its liver bed occurs often. However, connection is most likely to be established with the pylorus, the duodenum, and the colon. Invasion of the bladder, kidney, uterus, or portal vein is least likely to occur. External perforation through the abdominal wall is less uncommon. Occasionally the cystic veins are involved. This gives rise to purulent phlebitis and general sepsis (pyemia, endocarditis, etc.).

Subphrenic abscess (p. 2033) sequential to cholecystitis is peculiar only in as much as gallstones may be expelled by coughing. Large stones which ulcerate into the duodenum may be responsible for *gallstone ileus.*

In mild forms of cholecystitis a policy of noninterference is permissible; in severe cases operative measures of relief are imperative. Cholecystectomy is preferable to cholecystostomy. In cholangitis, cholecystectomy should be supplemented by drainage of the hepatic or the common duct (Kehr[12]).

Cholangitis, like cholecystitis, may be severe or mild. So-called catarrhal jaundice is really a mild form of cholangitis (perhaps a slight pancreatitis). If the inflammatory process extends upward into the small bile ducts, the term *diffuse cholangitis* is employed. This is likely to be fatal unless drainage is established early in the process (Israel[42]).

Cases of this sort present a menacing picture (chills, jaundice, etc.). If the papilla suddenly becomes patent or the obstructing stone is

liberated, the symptoms disappear as though by magic. Occasionally the establishment of a spontaneous choledochoduodenal fistula leads to the same result. In most of the nonfatal cases, however, the condition becomes chronic, the patient presenting a picture not unlike that of malaria.

It is to be remembered that in this class of cases the stone is also only the "opportunity thief" and that the pernicious causative factor is infective in character. So-called obstruction of the common duct is really a serous or seropurulent choledochitis. The inflammation subsides as soon as the papilla becomes patent, or the stone passes into the duodenum or slips back into the common or hepatic duct. The persistent presence of stone causes retention of bile, and the duct gradually dilates and thickens, sometimes to an enormous extent. Repeated attacks of inflammation impel the stone toward the duodenum and each attack is attended with transient incarceration associated with fever and jaundice. In many cases the patient develops an ashen gray color characteristic of chronic cholangitis. Prolongation of the process is responsible for biliary cirrhosis and cholemic diathesis, enlarged spleen, and ascites. Even in this stage of the affliction operative measures are likely to afford relief.

The *surgical treatment* of cholangitis consists in *drainage of the biliary passages.* In cases in which the general condition of the patient precludes employment of narcosis, cholecystostomy must, *per force,* be employed. It accomplishes the purpose in some cases. However, incision and drainage of the common duct (*choledochostomy*) and simultaneous cholecystectomy is the measure of choice.

Idiopathic cystic choledochus is a rare, congenital weakness of the wall of the duct with secondary valve formation (Dreesmann[43]). The condition occurs mostly in young girls. It is evinced by gradual enlargement of the right abdomen by a fluctuating mass and by intermittent jaundice. Kehr[12] advises choledochoduodenostomy as a measure of relief.

Tuberculosis and actinomycosis of the gallbladder are rare affections. They are occasionally uncovered in the course of operative procedures aimed at the relief of chronic cholecystitis and choledochitis.

Gallstone disease occurs most often in women of middle age. However, no age is exempt from the affliction. The causative influence of pregnancy seems to be established (Mayo[44]).

In the vast majority of instances gallstones develop in the gallbladder, though they also occasionally originate in the ducts. Their

development is favored by all factors which tend to interfere with the normal rate of flow of bile, i. e., by tight corsets, pregnancy, costal type of respiration, sedentary habits, etc.

Summarizing, one may say that the development of radial cholesterin stones in sterile bile may be explained on the basis of a cholesterin diathesis; in all other instances *the important causative factors* are biliary stasis and infection. In connection with the latter, the work of Welch[45] is particularly instructive.

The *pathological anatomy* of the various forms of cholecystitis and

FIG. 935, *A*.—INFECTED, MUCH THICKENED GALLBLADDER; STONE IMPACTED IN HARTMANN'S POUCH.

cholangitis has already been spoken of. It is certain that in the main, the character and virulence of the infection determines the severity of the inflammatory process and the changes in the biliary passages. The presence of stone in the neck of the gallbladder or in the supraduodenal portion of the duct simply provokes variations in the character of pathological changes in a mechanical sense. This is true especially when the stone is movable and varies in its location. Thus, it may be responsible for obstruction of the cystic duct and consequent distention of the gallbladder, or it may cause dilatation of the ducts, or, as it migrates, may cause all of these conditions.

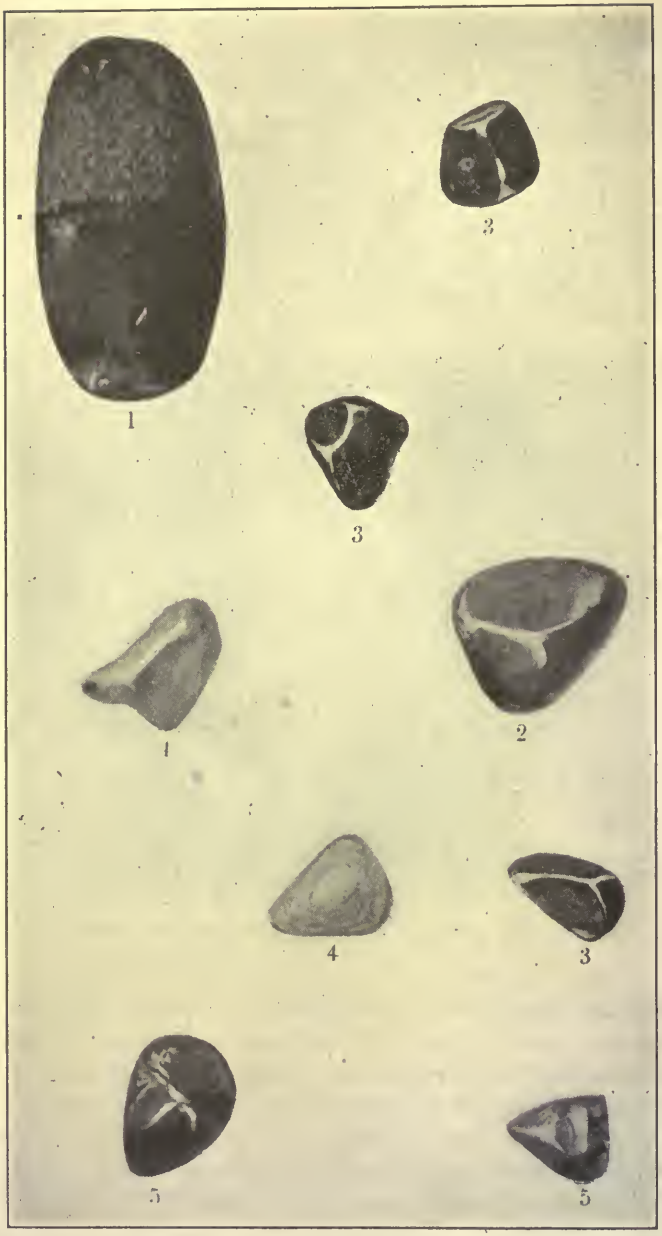

FIG. 935, *B*.— GALLSTONES OF VARIOUS TYPES.

1, Large symmetrical stone; 2, usual form of stone; 3, deeply pigmented stones; 4, section of stone showing lamination; 5, light brown stones.

The *symptoms and diagnosis* of cholelithiasis are best based on the findings of those experienced in this connection. This has led to separation of the problem into stone carriers and those suffering from gallstone disease, i. e., *latent and manifest cholelithiasis.* The oftener the surgeon operates in this class of cases, the more convinced he is, first, that the text book symptoms of cholelithiasis — colic, jaundice, enlargement of the liver, tumor of the gallbladder, etc.— are not constantly present; and second, that the diagnosis of gallstones is secondary in importance to recognition of the inflammatory process involving the biliary passages, though be it said that the latter is often associated with gallstones.

Except when visualized on the Röntgenographic negative (which is rare), the actual diagnosis of gallstones is possible only when concretions are vomited, expelled by coughing or are found in the feces. In the vast majority of cases the stones remain in the gallbladder, where they may, or may not, give rise to disturbances.

It is also true that the *latent stage* of cholelithiasis is not susceptible of recognition. Although Moynihan[46] has held that gallstones always produce symptoms, Ashoff[41] has shown that the radial cholesterin concretion is abacterial in origin and is not responsible for any disturbances. Kehr[12] says "We must insist that there is such a thing as a latent stage of cholelithiasis and that the latter need not give rise to symptoms. Under normal conditions the bile in the gallbladder is sterile and when stasis occurs the excitants of infection take advantage of the situation and invade the tissues of the organ. This is followed by bacterial cholecystitis." This is a view the Mayos[44] are inclined to take.

Recognition of the behavior of cholelithiasis is obtained in three ways:

1. By careful consideration of the previous course of the disease.
2. By thorough observation at the bedside.
3. By daily examination of the patient.

The Mayos[44] are insistent that the previous history of the case gives the most valuable information as to the character of the pathological process — a belief the writer regards as well founded. In many cases the diagnosis is established in this way. The character of the colic, occasional attacks of fever, intermittent, slight jaundice indicate the degree of the pathological process. Exact observation, in many instances, enables the surgeon to mentally visualize the condition of the biliary passages in a manner subsequently verified during operation. The

examination begins with inspection, which. in cases of large hydrops of the gallbladder, often reveals a dome-like protrusion at the upper, outer quadrant of the abdomen, and, when the patient is thin, the tumor may be seen moving with each motion of the diaphragm. *Percussion* informs us whether the distended gallbladder is a part of the liver. *Auscultation* is of little value. The friction murmur, generally regarded as indicative of pericholecystitis, is rarely discernible.

Palpation is of great diagnostic aid. Its value is much enhanced by experience. If the history of the case points to a gallbladder lesion, its area is not immediately "pounced upon," but the searching hand glides first over the lower quadrants of the abdomen and finally rests over the right hypochondrium, noting the various impressions gathered *en route*. The examination must be made gently and the abdominal wall relaxed by elevation of the patient's thighs; the knees are flexed. At the final goal, the left hand is placed against the right lumbar region and the two hands gently approximated. Previous colonic lavage is desirable. Hidden tenderness may be demonstrated by pressing both hands beneath the right costal arch, with the patient in the sitting position. The tenderness is best elicited if the patient arrests respiration in expiration (Murphy[47]).

Palpation gives information with regard to the position of the right lobe of the liver, the consistence and size of the entire organ, swelling of the gallbladder, etc.

When a Riedel's lobe is associated with hydrops of the gallbladder, it may be mistaken for movable kidney. Jaundice is usually attended with enlargement of the liver. However, the common duct is not always occluded. Swelling of the gallbladder without icterus indicates cholecystitis; its enlargement with icterus points to obstruction of the outflow of bile through the duct by a pancreatic tumor or pancreatitis. Pain in the region of the gallbladder argues for cholecystitis, though this has its exceptions. Severe pain suggests empyema of the gallbladder. Persistently elicited tenderness in the right hypochondrium bespeaks chronic ulcerative cholecystitis. An enlarged gallbladder can be made to disappear, but always returns to its position (a sort of ballottement). This is not the case with movable kidney. Artificial distention of the colon effaces the movable kidney.

In a general way one may say that cholecystitis with swelling is easily diagnosticated. Verification of the diagnosis by puncture is no more permissible than it is in abscess of the liver. When a tumor is

not palpable, errors arise in connection with lead colic, kidney colic, and intestinal colic.

In doubtful cases a study of the gastric functions, the urine and feces, gives valuable aid.

The diagnosis of acute obstruction of the common duct, after what has been said, is so evident that it need not be farther discussed here.

Chronic obstruction of the bile duct, however, frequently escapes recognition. Jaundice is absent when the duct is dilated, when the infection subsides, when the bile flows onward by the side of a stone, and when the cystic duct is patent and the bile gains access to the gut through a fistulous communication between it and the gallbladder. Colic is not always present and is often replaced by discomfort in the epigastric region. Frequent chills (every few days) attended with increase of jaundice suggest obstruction of the bile duct. It is important to determine whether the obstruction is due to stone or to extrinsic pressure. In chronic obstruction due to stone, the gallbladder is usually shrunken and not palpable; jaundice is slight and variable; the stool is alternately brown or gray; colic is not persistent; and fever is intermittent. In chronic obstruction by a tumor, the gallbladder is usually enlarged and palpable; jaundice is definite and progressively deepened; the stool is persistently gray; colic and fever are usually absent. This rule, laid down by Courvoisier,[40] is conformed to in 80 per cent of the cases.

A comprehensive view of the kaleidoscopic picture presented by cholelithiasis would seem to indicate that of the cardinal symptoms — icterus, swelling of the liver, and colic — the first two are almost always absent in inflammations restricted to the gallbladder and are observed only in conjunction with the third, in cholangitis. However, *colic* may be absent in severe cases of cholangitis, which may cause only indefinite discomfort referred to the epigastric region. As a matter of fact, pain in cholelithiasis is an illusive factor. Genuine colic appears most often in the form of gastralgia. It is frequently described as pressing, boring or stabbing in character and is referred to the right costal arch; not uncommonly it is characterized by a girdle pain, as lumbago, and occasionally, as a pain in the thorax. In the last instance it is, in elderly persons, likely to be mistaken for angina.

The third symptom — *jaundice* — is regarded by many as the most important symptom of gallstones, yet it almost never appears in cholecystitis. Icterus is usually an indication of simultaneous pan-

creatitis (Arnsperger,[48] Opie[49]); it is absent in 20 per cent of cases of stone in the common duct (Kehr[12]).

Mechanical icterus (cholelithiasis, pancreatic tumor, etc.) must be differentiated from the functional (cirrhosis, etc.). Urobilin in the urine argues for functional icterus. Operation is contra-indicated. As mechanical icterus may merge into the functional (biliary cirrhosis), operation must not be delayed.

Basing his classification upon that of Ashoff,[41] namely, (a) non-inflammatory gallstone disease, (b) inflammatory gallstone disease, Kehr[12] arranges the stages of the disease and its treatment as follows:

(a) In *non-inflammatory cholelithiasis* the stasis of bile follows a latent course and does not give rise to symptoms. It is attended with the development of abacterial radial cholesterin concretions. The cholesterin diathesis is combated by appropriate medication.

(b) *Inflammatory cholelithiasis* is dependent upon the formation of cholesterin stones, combination stone, and cholesterin-pigment-calcium stones provoked by infection.

INFLAMMATIONS OF THE GALLBLADDER

Acute Inflammations — Serous Cholecystitis.— The *symptoms* are mild colic (gastralgia), slightly tender gallbladder, tumor, no jaundice, no enlargement of the liver, little or no fever. Stones are not passed. The *treatment* consists in the administration of morphia, rest in bed, and attention to diet. Operation is not indicated.

Seropurulent Colecystitis.— The *symptoms* are the same as in serous cholecystitis, only more marked. Fever is preceded by a chill; the gallbladder tumor is distinct and tender. Operative procedure is indicated, if the symptoms do not subside in two or three days.

Phlegmonous, Gangrenous Cholecystitis.— The *symptoms* mentioned above are very severe; slight icterus from inflammation of the contiguous liver tissue; swelling of the glands in the hepatoduodenal ligament or those situated in the pancreas (Arnsperger[48]). The general condition is menacing. To this class must be added torsion of the gallbladder and necrosis (Mühsam[50]). The *treatment* consists in immediate operation. Rupture of the gallbladder and peritonitis are likely to occur.

All of these forms of inflammation may undergo spontaneous regression. However, this is probably only apparent. Once the gallbladder is damaged, the infection never entirely subsides, but persists in a chronic form.

Chronic Inflammations.— The *symptoms* of chronic cholecystitis depend upon the virulence of the invading bacterial excitants, upon the condition of the walls of the gallbladder (ulcerations, scars, intramural abscesses), upon the contents of the gallbladder (bile, serum, pus), and upon the size and form of the gallbladder, etc. Gallbladder tumor may be absent when the organ is shrunken. Jaundice occurs only when cholangitis is also present. The region of the gallbladder is almost always tender. Pain is no longer colicky, but becomes dull. The *treatment* consists in operation when the process does not subside under a general regimen.

INFLAMMATIONS OF THE BILE DUCT

Acute Inflammation.— Acute inflammation (choledochitis acuta) is usually called acute biliary obstruction. The *symptoms* are jaundice, colic, enlargement of the liver, and tenderness in the region of the gallbladder and in the median line. Healing usually occurs after passage of the stone. The *treatment* consists in the administration of morphin, diet, and gastric lavage. If the obstruction persists, operative relief must be considered.

Chronic Inflammation (chronic obstruction of the duct).— If the stone is located in the retroduodenal portion of the duct, icterus is deep; if in the supraduodenal part, jaundice is variable (the stone is movable); pain is colicky or dull. Fever and chills occur at intervals. The gallbladder is usually small (80 per cent), and the liver ultimately becomes cirrhotic. At first the urine contains only bilirubin, later urobilin (cholemic diathesis). Operation is feasible two months after the beginning of jaundice.

If the obstruction is due to a tumor's pressing on the papilla (pancreatitis, carcinoma of the pancreas), icterus appears without pain and steadily increases. Fever and chills occur; the gallbladder is large. The *treatment* consists in anastomosis.

THE REMOTE EFFECTS OF INFLAMMATION OF THE BILIARY PASSAGES ON CONTIGUOUS AND DISTAL ORGANS

1. *Liver:* Abscess, cirrhosis (cholemia), pylephlebitis purulenta.
2. *Stomach and gut:* Atony, pyloric stenosis, fistulae, obstipation, ileus.
3. *Peritoneum:* Peritonitis, subphrenic and retroperitoneal abscesses.

4. *Pancreas:* Acute and chronic pancreatitis, necrosis of the pancreas.

5. *Lung and heart:* Bronchitis, pneumonitis and pleuritis, myocarditis, endocarditis.

The much discussed question of "internal medication or operation" is answered by the special diagnosis. As long as the physician is satisfied with the simple diagnosis of gallstones, this question will remain unanswered. When, however, he learns to differentiate gangrenous from serous cholecystitis, and has an intelligent conception of the conditions obtaining in connection with chronic choledocholithiasis, he will know which patients to send to the "springs" and which to send to the surgeon (Kehr[12]). While errors in diagnosis will occur, there is no doubt that an accurate diagnosis is feasible in most cases.

The number of conditions that have to be taken into account in the differential diagnosis is large. The most important are 1 gastric and duodenal ulcers; 2 tumors of the pylorus; 3 tumors of the liver (carcinoma, hydatid cyst, hepatic lues, hepatoptosis); 4 movable kidney, hydronephrosis, cystic kidney, nephrolithiasis; 5 appendicitis, ileus; 6 carcinoma of the transverse colon; 7 pancreatitis, pancreatic necrosis; 8 hematoma of the abdominal wall; 9 gastric crises; 10 hysteria, neurasthenia.

In men, duodenal ulcer, and in women, hysteria, must always be excluded.

THE INDICATIONS FOR OPERATIVE TREATMENT OF CHOLELITHIASIS

Medical Treatment.— So-called medical treatment of gallstones does not accomplish anything with regard to the stones themselves. It is, however, capable of lessening the infection, and thus the disease passes into the latent stage. This succeeds in 80 per cent of the cases. The latency does not mean complete healing, but the patient is relieved and this is "as good as a cure." The other 20 per cent must be subjected to operative relief (1) because of an *absolute indication* in cases of gangrenous and seropurulent gallbladder, chronic cholangitis, acute severe cholangitis, perforation of the gallbladder, and beginning carcinoma; (2) because of a *relative indication* in cases in which the general health of the patient is in danger of permanent damage.

From a *pathological standpoint* this includes the cases of chronic ulcerative cholecystitis, infectious hydrops, and adhesive pericholecys-

titis; *clinically,* those which are not free from symptoms between attacks.

While operation is urged in all the cases in the category of the absolute indication, its execution in the others may be more or less influenced by the patient's viewpoint.

Early Operative Measures.— Early operative measures are not permissible 1 because the disease shows a great tendency toward latency; 2 because, compared with appendicitis, the dangers of the operation are not slight; 3 because an early operation to afford relief must consist in cholecystectomy and T-drainage of the duct in order to relieve the situation permanently. A precautionary operation with a mortality of 3 per cent is too dangerous.

The contra-indications as regards operation are similar to those obtaining in connection with all major operations.

Operative Method.— The question of method of procedure in cholelithiasis may now be definitely answered as follows: Cholecystectomy is preferable to cholecystostomy; choleeystendysis should not be employed at all; hepatic drainage should take the place of choledochotomy with suture.

The quarrel in this connection was easily settled when the surgeons agreed upon the indications for operation. Employment of cholecystostomy is permissible only in an emergency. When cholecystectomy is necessary, it should always be combined with hepatic drainage.

Cholecystectomy is more dangerous than cholecystostomy. The latter is followed by recurrence of the symptoms in from 18 to 20 per cent of the cases.

The disadvantages of choleeystostomy may be stated as follows:

1. An apparently normal gallbladder will often be found on section to be the seat of pathological changes.
2. Small stones are readily hidden in the folds of the cystic duct and are not revealed until the latter is removed with the gallbladder.
3. The gallbladder obstructs the search for stones in the common and hepatic ducts.
4. Gallstones originate almost exclusively in the gallbladder; if the organ is left *in situ,* recurrence is likely.
5. Cholecystostomy is likely to be followed by adherence of the gallbladder to the abdominal wall (even when the Mayo[44] tube drainage is employed) and becomes responsible for subsequent discomfort.
6. Preservation of the gallbladder has been followed by carcinoma (Kehr[12]).

7. The possibility of the establishment of biliary and mucous fistulae is greater with cholecystostomy than with cholecystectomy.

8. In cholecystostomy abscess of the liver is likely to be overlooked.

9. The average time required for healing after cholecystostomy is forty-nine days; after cholecystectomy it is thirty-one days (Ekehorn[51]).

Realization of the disadvantages of cholecystendysis and cholecystostomy has gradually caused the surgeon to perform cholecystectomy, which, however, when made alone, does not accomplish radical healing. Not until the combination of revision of the common duct and subsequent hepatic drainage was practiced did the gallstone operation achieve its greatest possibilities.

Weighing the advantages of hepatic drainage against those of choledochotomy and suture for comparison leads to the following:

Choledochotomy makes no provision for the relief of the infection almost constantly present in gallstone disease. Natural drainage is insufficient, because of the swelling of the mucosa of the papilla and the compression caused by the head of the pancreas. On the other hand, hepatic drainage furnishes egress to the infected bile. Consequently:

1. The mortality after choledochotomy with suture is much greater than when drainage is provided.

2. Choledochotomy closes the duct so that it cannot be lavaged. Stones are left *in situ* in 20 per cent of the cases. Hepatic drainage makes possible subsequent expulsion of stones. Even then stones remain *in situ* in 2 per cent of the cases.

3. In choledochotomy, healing *may* take place in fourteen days. This advantage is illusionary, on the ground that in cases in which suture is permissible, hepatic drainage is also followed by healing, i. e., external discharge of bile, in from fourteen to twenty days.

4. Efforts at extraction of stones provoke lesions of the mucosa, which bleed heavily into the duct. This is especially dangerous in cholemia. Hepatic drainage conduces to greater control of the bleeding than choledochotomy with suture.

5. When proper tamponade is employed in connection with hepatic drainage, peritonitis is practically impossible.

6. The external discharge of bile in hepatic drainage may lead to serious nutritional impairment. When T-drainage is employed this is obviated.

7. Hepatic drainage is technically easier than choledochotomy and "bile tight" suture.

8. Hepatic drainage makes sounding of the papilla possible.

All these facts would seem to justify abandonment of choledochotomy with suture as a measure of relief in gallstone disease.

The *prognosis* in cholelithiasis, on the whole, is good. In manifest cholelithiasis the outlook is not so favorable. Serous cholecystitis usually runs its course without danger to life. The chief menace in the disease lies in cholangitis, cholemia, infection, and carcinoma. In uncomplicated cases the persistence of disturbances may render the patient incapable of earning a living. The tendency to complete healing is slight. However, mergence into latency is frequent. Spontaneous expulsion of stones *per vias naturales* may be followed by healing, but this is uncommon. Usually enough stones remain *in situ* to reprovoke inflammation. Expulsion of stones through fistulous openings into hollow organs may also be followed by healing. However, the adventitious opening affords opportunity for the invasion of bacteria into the bile passages, and chronic cholangitis follows.

Harmless as the latent stage is, manifest cholelithiasis is attended with many dangers. Of these, acute and chronic empyema and chronic biliary obstruction with fever, chills, and icterus offer the most unfavorable prognosis. In these cases the dangers of operative relief are less than obtain with a policy of expectancy. In diffuse purulent cholangitis surgical treatment holds out the possibility of recovery (Israel[42]). The prognosis of cholelithiasis cannot be based on the disease as a whole; too many factors must be taken into account in each case to permit this. While one is not, as in appendicitis, justified in operating as soon as the diagnosis is made, surgical measures of relief employed at the right time certainly obviate a fatal outcome in cases of severe infection of the biliary passages.

At this writing operative measures of relief are aimed, not only at the gallbladder but, in most cases, also at the bile ducts. This means that, if any method of selection is permissible, cholecystectomy in combination with hepatic drainage is the procedure of choice. This is, therefore, described first.

While it is impossible to lay out a definite scheme of procedure applicable to each case, it would, nevertheless, seem permissible, from a pathologico-anatomical and clinical standpoint, to divide the problem into two groups which demand definite technic:

1. The gall bladder is enlarged and inflamed; it contains stones; the common duct seems to be patent; icterus is absent.

2. The gallbladder is small and shrunken and is free from stones, while the common duct is filled with concretions; icterus is present.

In all operations on the gallbladder and biliary passages a considerable advantage is derived from the use of a sandbag or a cushion placed under the patient's back at, or a little above, the level of the

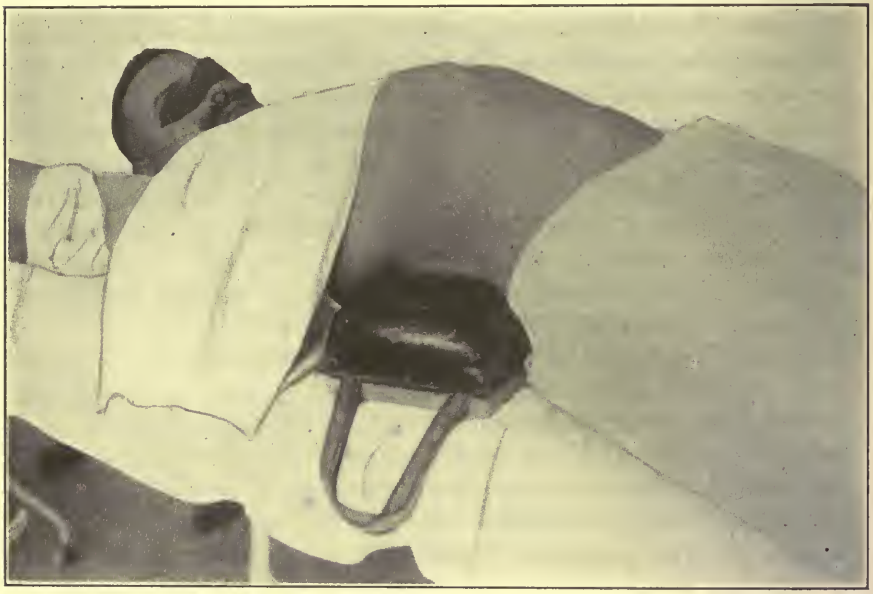

FIG. 936.—POSITION OF PATIENT FOR OPERATIONS UPON THE BILIARY PASSAGES (Kehr).

liver. Modern operating tables are arranged so that this position is easily attainable (Fig. 70).

The incision has been made the subject of much discussion. In this connection Moynihan[52] says "The best incision is a vertical one made at first about 4 to 5 inches in length through the right rectus near its outer border. The upper end of the incision starts at the costal margin and extends vertically downwards. If more room is needed than this incision gives, it may be obtained by prolonging the incision downwards, or by carrying the upper end obliquely upwards and inwards, dividing the fibers of the rectus about ½ inch from the costal margin. There is rarely any need for a further increase of the

incision than these. The incision along the outer margin of the rectus, with the upward and inward extension, is that first suggested by Mayo Robson.[53] Great convenience may often be gained, especially in stout patients, with an abdominal wall 3 inches or more in thickness, by making the skin incision 2 or 3 inches longer than the incision in the rectus. The sides of the wound then fall away and allow the more ready access of the hand. The longer incision in the skin and subcutaneous fat does not, in any way, weaken the abdominal wall, as a longer incision in the muscles would certainly do. Kocher[54] uses an oblique incision 4 inches in length, about 1½ inches below the costal margin. The center of the incision is a little outside the outer margin of the rectus muscle. This is a very useful incision, giving ready

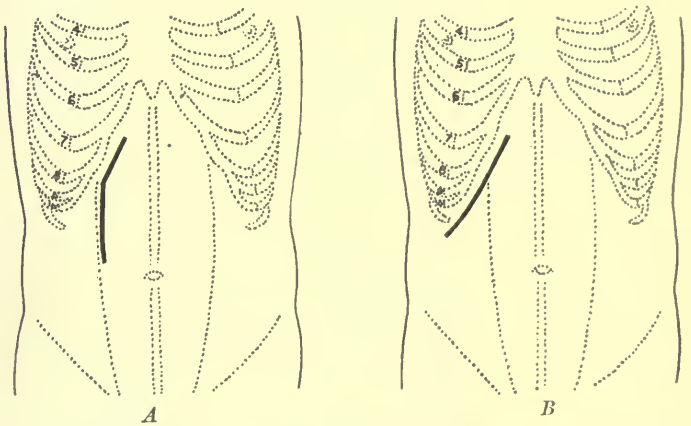

FIG. 937.— *A*, MAYO-ROBSON'S INCISION. *B*, KOCHER'S INCISION.

access to the gall-bladder and ducts, being readily enlarged either inwards or outwards, and doing little damage to the nerves and muscles of the abdominal wall. Very little weakness of the parietes remains after the operation and there is little chance of the development of a hernia. This incision and the vertical incision, with Mayo Robson's extension, are the only ones I have adopted. So far as I am aware, I have not had a single case of postoperative hernia. This I attribute in part to the method of making the incision (a large skin wound and a small muscle wound), but chiefly to care in stitching up the wound. Kehr[12] makes use of a very extensive incision which he calls the 'wellenschnitt.'

"However, with a vertical incision 5 or 6 inches in length, and at

the most, an oblique upward and inward prolongation of this just below the costal margin, any operation can be performed upon any part of the gallbladder or the cystic or common or hepatic ducts, provided the ducts are brought within easy reach; then the smaller the incision the better, for the intestine can more readily be packed away with swabs. A long incision is troublesome, in that it allows the escape of intestines from the wound and makes the retention of the bowel within the abdomen a matter of constant attention."

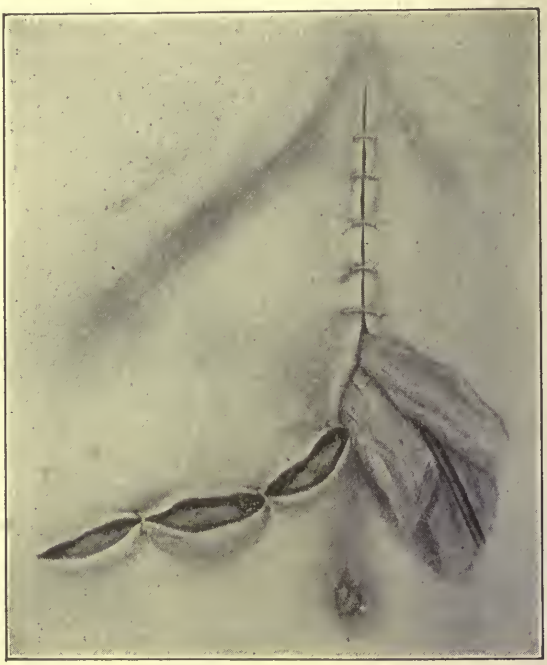

FIG. 938.—KEHR'S INCISION FOR APPROACHING THE BILIARY PASSAGES; GAUZE TAMPONADE; TUBE DRAIN LEADING FROM CHOLEDOCHUS (Kehr).

On the other hand, there is no doubt that Kehr's incision conserves the nerve supply of the rectus muscle, and that it gives the best exposure, not only for approaching the biliary passages, but also for providing access to the other organs (pylorus, duodenum etc.) which it may be necessary to attack in connection with the surgery of this region. The lower vertical prolongation of the incision need not be made unless the contingencies arising in a given case demand it.

Opening of the abdomen is followed by INSPECTION AND PALPATION OF THE GALLBLADDER, THE LIVER, AND THE CYSTIC DUCT. Adhesions must be separated with great care. In this way fistulae are often uncovered.

When the gallbladder is freed down to the cystic or common duct, it is, when distended, aspirated, and its excision is immediately pro-

ceeded with. The serosa, where it merges with the liver, is sectioned and the gallbladder is dissected from its bed in the liver. The cystic artery is tied separately at the neck of the organ. The gallbladder is

FIG. 939.—CHOLECYSTECTOMY.

The gallbladder dissected from its bed; the cystic artery being divided separately (Kehr).

then easily pedunculated, and the cysticus is sectioned at the choledochus, after it has been clamped to prevent escape of its contents.

In isolating the cystic duct one must remember that its junction with the bile duct is exceedingly variable. It may be at an acute angle, or may run parallel with the hepaticus, or it may partially en-

circle the choledochus. This argues against removal of the gallbladder from the duct outward, which involves danger of injury to the latter during the manipulation.

The choledochus is always probed through the stump of the cysticus. When the bile is clear, when the history of the case does not indicate the presence of obstruction or of cholangitis, when the pancreas is soft and the choledochus is not dilated, one may be content with simple cholecystectomy. Otherwise, the common duct must be incised on a sound introduced into it. In doing this the gastroduodenal artery, which crosses the duct just above the duodenum, must be avoided. The edges of the wound in the duct are grasped with clamps, stones are removed, and the duct is drained with a T-tube; the latter is sutured in place. The wound of approach is drained with gauze (cigarette drain).

In the second type of case, chronic icterus is the outcome of chronic cholangitis, and this is maintained by the presence of a large stone in the common duct, usually located above the duodenum. In 80 per cent of these cases the gallbladder is shrunken, and the surgeon may attack the common duct at once. The duct is isolated, as stated above, opened over the stone, and drained by means of a T-tube. The gallbladder is removed last.

In two places the removal of stones is difficult, when they are located high in the hepatic duct above the bifurcation, and when they lie behind the duodenum.

Stones in the hepaticus are removed through a separate incision (hepaticotomy). In doing this the cystic artery and the right branch of the hepatic artery must be taken into account.

Retroduodenal stones may be removed by mobilizing the duodenum (Kocher[54]). Very often the stone can be "milked" up into an accessible portion of the duct. If this does not succeed, the problem is attacked via the retroduodenal or transduodenal route. Each method has its lights and shadows. The writer prefers the transduodenal route. For the purpose, the anterior wall of the duodenum is opened *transversely,* the stone is pressed out of the papilla, or the latter is split and the stone extracted. If the stone is impacted in the ampulla, the posterior wall of the duodenum is opened, the duct is split, and the stone is removed. The edges of the wound in the duct are sutured to the edges of the duodenal incision (choledochoduodenostomy of Kocher[54]). In both the retroduodenal and the transduodenal methods the hepaticus is drained through the supraduodenal incision.

Radical cholecystectomy is evolved from the conservative methods — cholecystendysis, cholecystotomy and cholecystostomy. All these have been forced into the background.

Cholecystostomy consists in opening and draining the gallbladder.

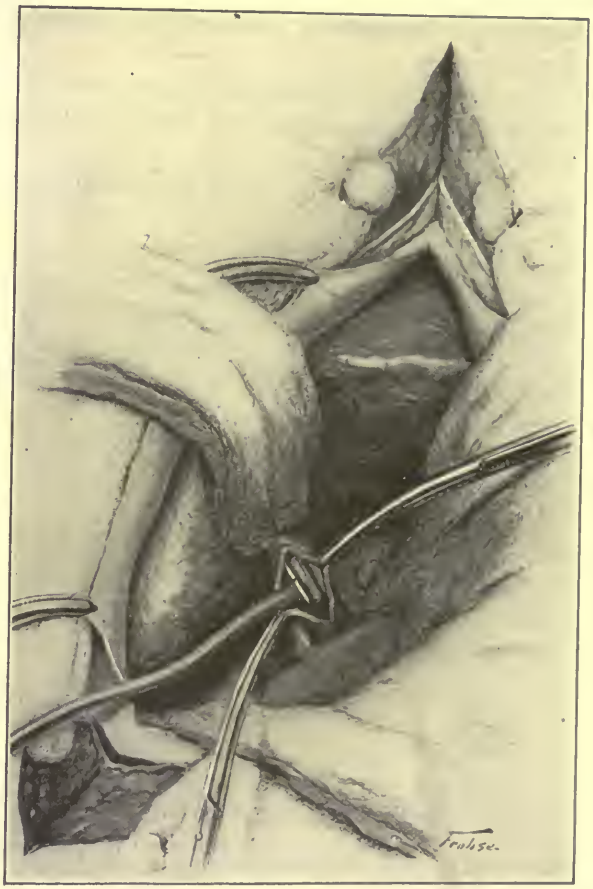

FIG. 940.— DRAINING THE CHOLEDOCHUS.

The liver is turned up into the wound. The gallbladder is removed. The T-drain is introduced into the choledochus (Kehr).

At this writing the operation is performed at one sitting. After the abdomen is opened, the gallbladder is aspirated and its walls are grasped with forceps. Through a small opening in the organ a drain-

age tube is introduced and the walls of the gallbladder are inverted over it by means of a purse string suture in the manner shown in Fig. 941. The operation is permissible in cases of emergency.

Cholecystdochotomy consists in incision of the cystic duct and removal of a stone impacted in it. If done at all, it must be performed in conjunction with cholecystostomy. Cholecystdochotomy and suture is a daring maneuver.

The ideal operation of *cholecystendysis* (Courvoisier[40]), i. e., opening the gallbladder, removing stones, and suturing the organ, possesses only historical interest.

The part the pancreas plays in connection with cholelithiasis is taken up in chap. xv.

Anastomosis between the biliary passages and the gastro-intestinal canal is practiced in cases in which it is impossible to reëstablish the normal avenue of flow of bile. This is rarely, if ever necessary in cases of cholelithiasis. In 1,900 cases of this sort operated upon by Kehr,[12] anastomosis was not practiced once.

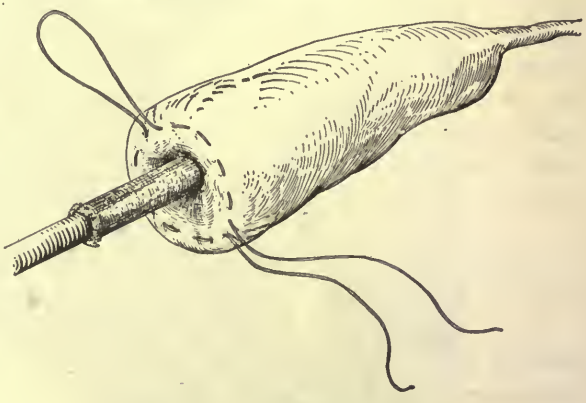

Fig. 941.— Cholecystostomy.
Application of pursestring suture around the tube.

In cases of retroduodenal stones v. Bardeleben[55] very properly regards an anastomosis as easier than the measures of relief indicated above (transduodenal choledochotomy, etc.). However, the former does not remove the foreign body and thus the element of infection is not eliminated.

In cases of cicatricial stenosis and of tumors which obstruct the hepaticus and the choledochus, anastomosis often becomes necessary.

If the obstruction is located at the hilus of the liver, in the hepaticus, or higher, anastomosis between the finer biliary ducts and the gut or stomach (hepaticocholango-enterostomy or gastrostomy) becomes a measure of necessity (Kehr,[12] Garré,[56] Erdmann[57]). In some cases it

is feasible to establish communication between the gallbladder and its bed, and then to anastomose the former to the gut or stomach (hepaticocholangocystenterostomy or gastrostomy).

When the obstruction is located at the point of entrance of the cysticus into the hepaticus, so that the bile flows neither into the gallbladder not into the choledochus, a hepaticocysto-enterostomy may be

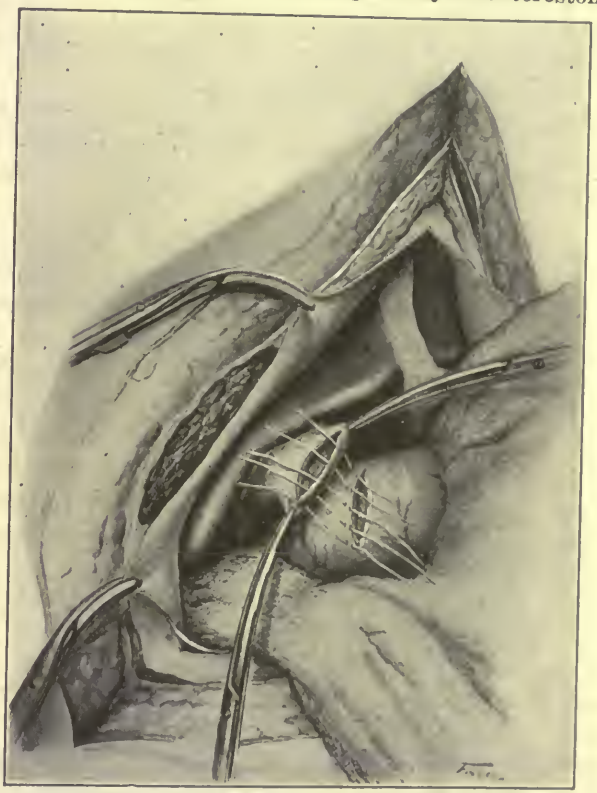

FIG. 942.—CHOLECYSTGASTROSTOMY BY SUTURE ONLY (Kehr).

made. When the hindrance is situated in the supraduodenal or retroduodenal part of the choledochus, a cholecystenterostomy or gastrostomy relieves the situation.

In retroduodenal occlusion it is occasionally feasible to establish direct communication between the choledochus and the duodenum (choledochoduodenostomy).

The technic of these procedures is in every regard similar to that

employed in connection with gastro-enterostomy. As the patient is always profoundly jaundiced and suffers from cholemia, it is necessary to precede the operation by the use of agents which increase the coagulability of the blood (gelatin, horse serum, etc.).

Haasler[58] has shown that a number of goodsized dilated bile ducts situated at the lower aspect of the left lobe of the liver are particularly well adapted to anastomosis. Therefore, it seems best to excise a wedge of liver tissue (6 cm. by 2 cm.) from this portion of the liver and attach it to the pylorus or stomach.

Of the various operations, *cholecystenterostomy* is the one more frequently employed. However, this is being rapidly replaced by anastomosis with the pylorus or the stomach (Hildebrand,[59] Kehr,[12] Erdmann[57]).

Tumors of the Gallbladder and Biliary Passages.— In the gallbladder, benign tumors (adenomata, myomata, fibromata, lipomata) occur less frequently than the malignant (carcinomata, sarcomata). Cystadenomata of the outer wall of the gallbladder are described by Kaufmann.[60] Aschoff and Bacmeister[41] describe adenomatous deposits (congenital origin) which are found in the fundus of the gallbladder and which may be responsible for carcinomata in this situaton. Other pathologists regard these processes as secondary to chronic inflammations. Papillary fibro-epitheliomata not infrequently develop in this situation. They are analogous to the papillomata found in the urinary bladder, which are also regarded as secondary to chronic inflammation and may be responsible for biliary stasis. Pels-Leusden[61] discusses the problem at great length.

Of the malignant processes involving the gallbladder, carcinoma occurs much more frequently than sarcoma.

Carcinoma of the gallbladder is seen in advanced life. The *diagnosis* is possible only when a nodular gallbladder is palpated. The process is often attended with empyema of the organ and is uncovered during surgical attack for relief of the latter. The suspected causative relationship between chronic cholecystitis and carcinoma argues for early operation in cases of the former in elderly persons.

The operative treatment is not satisfactory. However, resection of the process has been followed by protracted freedom from return of the disease (Körte,[62] Mayo,[44] Robson,[53] etc.).

Primary carcinoma of the biliary passages originates in the epithelium of the glands of the duct, most often in the papilla of the duo-

FIG. 943.— RESECTION OF THE CHOLEDOCHUS AND CHOLECYSTOTOMY.

Above, the gallbladder is delivered; the choledochus is clamped. Below, the choledochus has been sectioned and is being "rolled up" with the forceps. The cystic artery is tied. The dotted lines show where the hepaticus is sectioned (Kehr).

denum, at the entrance of the cystic into the hepatic duct, and at the bifurcation of the hepaticus. The scirrhous occurs more frequently than the medullary form of the disease. The gallbladder is usually enlarged; gallstones are rarely present. *Jaundice* is at first slight, but later becomes intense *Pain* is not a dominant symptom. Donati[63] has collected and analyzed 102 cases of carcinoma of the cystic, hepatic, and common ducts.

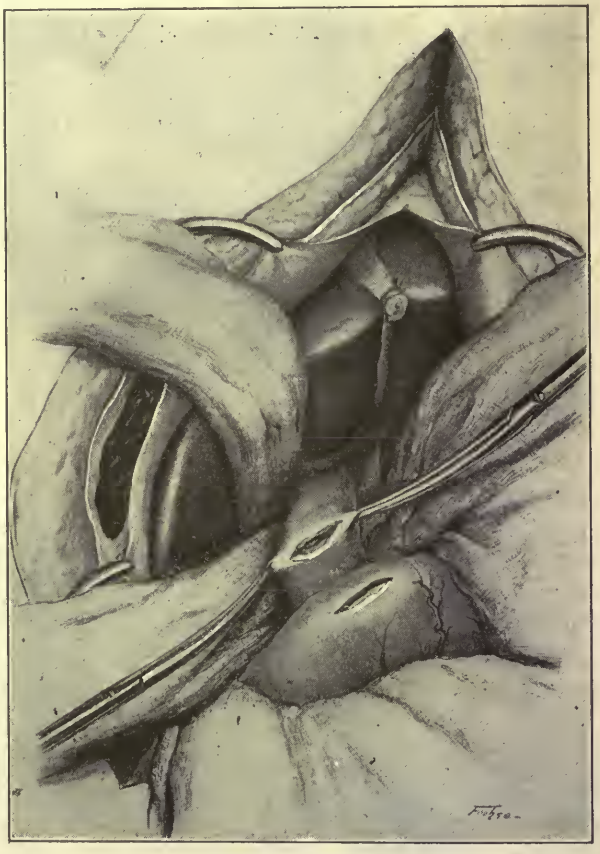

Fig. 944.— Hepaticogastrostomy (Kehr).

The *operative measures of relief* are directed toward resection of the involved passages and their end to end anastomosis. Should this fail, a palliative cholecdochgastrostomy or duodenostomy may be substituted. In a few instances it has been feasible to excise a portion of the common duct and to maintain the communication by means of a rubber tube. Spontaneous expulsion of the tube has (in several instances) been followed by cicatricial canalization and discharge of the tube into the duodenum. Successful operations of this sort are reported by Halstead,[64] Edes,[65] Mayo,[66] and Kehr.[12]

It is worth repeating that icterus of two months' standing may still be relieved by operation; the cholemia may be attended with irremediable conditions.

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CHAPTER XIV

SURGERY OF THE SPLEEN

General Considerations.— The province of the surgeon, as far as the spleen is concerned, is limited to its removal (*splenectomy*). It is, therefore, necessary that the effect of this step upon the organism be understood.

An extended discussion in this connection does not belong here. However, it is proper to state that the spleen is not necessary to life, that its removal is not attended with objective or subjective disturbances, and that the splenectomized patient is not hampered in the functions of life.

The following changes occur in the blood:

1. The *number of erythrocytes,* as a rule, is not affected and soon reaches the normal. In some instances there is a definite increase in red cells (hyperglobulia), ascribable to hyperfunction of the marrow of the bones.

2. The *formation of hemoglobin* is not impaired. On the contrary, splenectomy executed upon anemic individuals is followed by an increase in the hemoglobin content of the blood.

3. The total quantity of *white corpuscles* is at first increased; later it recedes to the normal. There is, however, a relative increase in lymphocytes and eosinophils.

4. Very often several groups of *lymph nodes* are transiently enlarged. This is regarded as vicarious and accounts for the lymphocytosis. Occasionally, indefinite, dull pain is experienced in the long bones.

As a whole, one may say that the function of the spleen is not understood. The view of Brogsitter,[1] who is inclined to believe that the organ acts as a reservoir ready to take care of unusual conditions, is not irrational.

The *new formation* of spleen tissue in splenectomized patients has been observed. V. Stubenrauch,[2] who celiotomized patients whose spleen had been removed years before, found the peritoneum studded

with small tumors consisting of spleen tissue. Heinecke[3] suggests that the tumors may have been so-called blood lymph glands.

The assumption that removal of the spleen is followed by *lessened resistance* to infection is not proved as far as the human is concerned.

Splenectomy.— The *spleen is best exposed* by an oblique incision beginning 2 cm. below the ensiform cartilage, dividing the rectus and extending a thumb's breadth below the edges of the arch of the ribs to the *axillary line.* A vertical transrectus incision does not give sufficient exposure and interferes with the innervation of the muscle. In the event of the presence of adhesions, *resection of the costal arch* may be necessary. The cartilages are readily divided through the oblique incision and the flap turned upward (Lejars[4]).

Transpleural exposure of the spleen is not a difficult problem. See chapter on Surgery of the Thorax.

In *extirpation of the spleen* the hand readily delivers the organ, and the vessels at the hilum, together with the gastrosplenic ligament, are tied and the pedicle is cut. If the spleen is very large, it may be necessary to ligature the pedicle in sections. If adhesions are present. they are carefully divided between ligatures.

Suture of the spleen, following injury, for instance after implantation of the thyroid tissue (Payr[5]), may be necessary. The friability of spleen tissue makes this troublesome. Danielsen[6] uses fine round needles and merely closes the capsule. Most surgeons use round needles and chain sutures (Heinecke[3]).

Malformation of the spleen possesses little surgical importance (Willis[7]).

Injuries of the Spleen.— The *healthy spleen* is rarely injured, while the enlarged organ, which extends far below the arch of the ribs, is not infrequently the victim of trauma. Of the diseased spleens, the *malarial* is most often ruptured.

Injuries of the normal spleen occur most often in men. Open injuries are less frequent than the subcutaneous.

Contusion of the spleen, i. e., without rupture of the capsule, usually escapes recognition. It may be responsible for the formation of a blood cyst.

Subcutaneous rupture of the spleen is of importance. It usually occurs in connection with direct force, i. e.. a blow, a kick, buffer crushing, etc.

The *form and extent of the rupture* vary widely. The organ may be converted into a pulpy mass, or the tear may be limited to the cap-

sule (*ruptures sèches de Catteloup*). Occasionally, the organ is entirely avulsed from its pedicle. At times the spleen pulp is crushed and the capsule remains intact; finally the capsule ruptures (secondary rupture).

The immediate effect of rupture of the spleen consists in *hemorrhage into the abdominal cavity.* At first the blood collects in the subdiaphragmatic space, and later slowly extends into the general cavity. The rapidity with which this occurs accords with the severity of the lesion. Profound initial shock may be attended with transient arrest of the bleeding.

Spontaneous healing of a ruptured spleen is rare; most cases die of hemorrhage.

The *clinical picture* of subcutaneous rupture of the spleen is that of injury of an intraperitoneal organ with bleeding. Shock is usually severe and in most cases merges with the symptoms of hemorrhage. Pain is severe; it is at first located in the left hypochondrium, but soon spreads over the abdomen and into the left leg.

Examination of the abdomen reveals a *board-like rigidity.* The belly is flat, and the muscles are in a state of tetanic contraction. The upper abdomen is especially tender. The cremaster muscle is contracted, the testicle, drawn upward, and the penis, slightly erect ("like after a cold bath"—Trendelenburg[8]). Meteorism is not present at first. The effusion of blood in the abdomen is usually readily recognized by percussion.

In *secondary rupture* there is a reaction from the primary shock followed by the picture just described any time from a few hours to fourteen days.

Open injuries of the spleen usually involve both the thorax and the abdomen. This is especially true of gunshot wounds. Occasionally the entire spleen is prolapsed through an extensive abdominal wound.

The *treatment* of rupture of the spleen consists in surgical measures of relief.

In doubtful cases a median celiotomy is indicated. If the spleen is found injured, the rectus is divided transversely. If the diagnosis is certain, the oblique incision is preferable.

When the spleen is exposed, three methods of procedure come under consideration, extirpation, suture, and tamponade. *Splenectomy* is the method of choice. Suture is permissible in incised, stab, and gunshot wounds and in small ruptures with smooth edges. Tamponade is restricted to desperate cases.

Injuries of the pathological spleen are characterized by the disproportion between the degree of trauma and the extent of the destruction of spleen tissue. In this class of cases the spleen may be ruptured by a light blow with the hand or a cane, by a fall on a flat surface, and even by muscular action, such as vomiting, sneezing, coughing, etc.

Strange to say, the large spleen of leukemia is comparatively rarely ruptured. This is true also with regard to the enlarged spleen of pseudoleukemia, the amyloid and the tuberculous spleen. Rupture of a *typhoid spleen* usually occurs in the third or fourth week. Those rupturing during a relapse are usually secondary to an hemorrhagic infarct.

By far the most frequent ruptures occur in connection with *malaria* in the chronic stage, though not a few are coincident with tropical splenomegalia (kala-azar).

The *treatment* is smilar to that of the healthy spleen.

Abscess of the Spleen.— Most abscesses of the spleen are embolic or metastatic in origin. They develop most frequently in connection with *septic processes,* and are not rare in the acute infectious diseases, especially *septic endocarditis.* Typhoid abscesses are by no means rare. A certain number are sequential to contusions. Küttner[9] presents some interesting statistics in this connection.

As a rule, the suppurative infection is primarily multiple and later the foci coalesce into one large abscess. Gradually the capsule is perforated and the contents of the abscess enters the peritoneum, where either a circumcribed or a general peritonitis is developed. In other instances the spleen becomes adherent to a contiguous organ, such as the stomach or gut, and perforates into it. *Subphrenic abscess* is a not uncommon complication.

The *clinical picture* of abscess of the spleen is likely to be insidious. It is attended with pain in the left hypochondrium, with the general symptoms of a septic process, and with the local manifestations of an inflammation. Leukocytosis is usually very high.

The *diagnosis* is not exactly easy. The previous existence of one of the causative diseases is helpful in arousing the supicion of the surgeon. Diagnostic aspiration is permissible. In the latter event, the conditions necessary to immediate operation must be provided.

The *treatment* consists in splenectomy, if this is not prevented by adhesions (perisplenitis). If extirpation is not feasible, the abscess

is drained. The oblique incision described above gives the best exposure.

Cysts of the Spleen.— Of the non-parasitic cysts of the spleen so-called *blood cysts* are the most common. They often reach enormous dimensions. In most instances the lesion is ascribable to *trauma* which is followed by an effusion of blood, and this later becomes cystic. The *contents* of the cyst consists of a chocolate colored fluid containing cholesterin and a large quantity of coagulated blood.

The cyst is usually located on the surface of the spleen and protrudes into the abdominal cavity. As time goes on, the cyst may occupy the entire left side of the abdomen. In some instances the cyst becomes adherent to the surrounding organs, in others, it simply displaces the latter.

Serous cysts of the spleen are rare. While their origin is obscure, it is probable that they are either blood cysts, the coloring matter of which has been absorbed, or consist of dilated lymph vessels.

The *clinical symptoms* of cysts of the spleen are those of a slowly increasing tumor in the region of the spleen, which causes pressure disturbances in connection with the contiguous organs. At first the lesion does not give rise to symptoms; later, digestive disturbances, obstipation, and radiating pain develop. *Examination* of the splenic area reveals the presence of a smooth sharply defined tumor protruding from beneath the left costal arch; the latter is often lifted. Fluctuation is usually discoverable. The Röntgenogram shows increased arching of the diaphragm.

The *diagnosis* is not difficult when the tumor is characteristic. In other instances, the process must be differentiated from uronephrosis and pancreatic cysts. A prolapsed cystic spleen closely resembles an ovarian cyst. The character of the cyst may be determined by aspiration.

The *treatment* consists in splenectomy.

Echinococcus of the Spleen.— Localization of the cyst worm in the spleen is rare. The cyst is usually solitary, but, as a rule, coexists with similar lesions in other portions of the body. It may develop to an enormous size and present a physical condition of affairs similar to that described in connection with blood cysts. Perforation into the abdominal cavity also occurs in accord with the activity of the process.

The *diagnosis* can be made with certainty only with the help of the aspirator. Occasionally the presence of friction on palpation indicates the nature of the lesion.

Suppuration of the cyst is a common complication. In this event, aspiration, except when the mass is fixed, is a dangerous procedure.

The *treatment* consists in splenectomy, unless suppuration has occurred in a still movable spleen. In this event, the infected cyst is first fastened to the abdominal wall and subsequently is opened and drained. When the cystic growth encroaches upon the thorax it is best attacked by the two stage transdiaphragmatic method.

Tumors of the Spleen.— Of the tumors of the spleen, primary *sarcoma* is the most common. The tumor appears in the form of a rapidly growing nodular enlargement of the spleen, which is at first movable and ultimately attached to the ribs (Simon[10]).

The *diagnosis* is made only by celiotomy. *Primary carcinoma* of the spleen does not exist.

Cavernous angioma occasionally grows sufficiently large to be recognized as a tumorous enlargement of the spleen (Theile[11]).

Tuberculosis of the Spleen.— While miliary tuberculosis of the spleen does not possess surgical interest, the *chronic caseous nodular* form is susceptible of surgical relief.

Tuberculois of the spleen is occasionally *primary.* However, in most instances, it occurs in connection with tuberculosis of the lungs, peritoneum, or mesenteric glands. This does not contra-indicate splenectomy any more than a similar condition of affairs acts as a deterrent as far as nephrectomy is concerned.

The *clinical picture* of tuberculosis of the spleen is not characteristic. It is attended with a tumorous nodular enlargement of the spleen with a blood picture which may indicate tuberculosis.

The *diagnosis* is made only after exposure of the organ. The local findings are similar to those found in Banti's disease and in tumors of various kinds (Bender[12]).

Splenectomy in Malaria.— Splenectomy in malaria does not affect the course of the disease. It is, however, justified when the spleen is so much enlarged that it makes pressure on contiguous organs or becomes adherent to them, so that its drag interferes with function. Occasionally, the drag on the pedicle of the enlarged spleen causes severe pain, especially when the former is twisted. Under these circumstances splenectomy is followed by relief which often converts a useless person into a wage earner (Jonnesco,[13] Michailowsky,[14] Solieri[15]).

Splenectomy for Relief of Leukemia and Pseudoleukemia.— This procedure belongs to history. It has no influence on either of these

diseases and is justified only on the basis taken up in connection with malarial spleen (Rochard,[16] Küttner[17]).

Splenectomy for Relief of Splenomegalia.— Splenectomy for relief of primary splenomegalia (idiopathic splenic hypertrophy, Banti's disease, splenic anemia, splenomegalia primitiva and Gaucher's disease) is followed by brilliant results.

The form of splenomegalia to which this applies is very likely to be associated with *cirrhosis of the liver* (Banti's disease). According to Banti,[18] the condition consists in a primary, characteristic, histological, splenic enlargement, probably of toxic or infectious origin, as the result of which the spleen gives off certain poisons which cause serious changes in the blood forming organs and the liver. If the spleen is extirpated early in the disease, the concomitant changes recede. The underlying cause of the disease is unknown.

The disease is divided into three stages. In the first (3–5–12 years), the spleen very slowly enlarges, while a gradually progressing anemia develops, and the patient becomes increasingly asthenic. In the second stage, the liver enlarges while the spleen remains stationary; the anemia steadily increases. The third stage is characterized by ascites, and the liver shrinks, much as it does in cirrhosis. Banti assumes that the splenic toxin produces the anemia, the cirrhosis, and the asthenia.

The *blood* shows a decrease in hemoglobin content, while the red corpuscles are increased, normal, or lessened in number; leukopenia is present. The histological changes are fully described by Banti.[18]

The results of splenectomy in the first two stages of the disease are very satisfactory. In the third, splenectomy combined with the Tálma-Morrison operation (epiplopexy) may be followed by recovery (Jaffe[19]).

Anemia splenica is probably identical with the first stage of Banti's disease (Carstens[20]).

Splenomegalia, as described by Gaucher,[21] is wrapped in darkness. It is characterized by a chronic swelling of the liver and spleen, with pigmentation of the skin, and leukopenia. Finally (after years), the spleen develops a tumorous condition, which Gaucher calls an endothelioma. Splenectomy is followed by improvement in the general condition of the patient (LeFort,[22] De Josselin de Jong and Siegenbeek van Henkelom[23]).

Movable Spleen.— Movable spleen is no doubt congenital in origin,

although it probably occurs also in connection with acquired *enteroptosis* (*splanchnoptosis*).

The movable spleen is displaced in the arc of a circle, the axis of which corresponds to its pedicle. It is at first freely movable, but later often becomes adherent to a more or less distant organ (uterus, adnexa, bladder) and is then really a dislocated spleen. The pedicle may be sufficiently twisted to cause strangulation.

The *treatment* consists in *splenopexy* or *splenectomy*. The latter is the procedure of choice (Aillaud,[24] d'Arcy-Power,[25] MacDonald,[26] Stierlin[27]).

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CHAPTER XV

THE SURGERY OF THE PANCREAS

Anatomical Considerations.— The pancreas is a narrow elongated gland, lying behind the peritoneum and stretching transversely across the posterior abdominal wall. With the exception of the tapered extremity, which is in relationship with the spleen, the greater part of the organ is situated in the epigastric region. For purposes of description the gland is divided into several parts. The flattened extremity which occupies the duodenal concavity, or head, is connected to the body of the organ by means of the neck, a narrow bridge of gland substance. The tapered extremity, which is in relation with the hilum of the spleen, is called the tail.

The ductus choledochus passes through the head and is in contact with the second portion of the duodenum. Helly[1] found that, in 37.5 per cent of the cases, the duct was placed in a groove occupying the posterior surface of the gland; in 62.5 per cent it was entirely surrounded by gland substance. The lower end of the duct is in contact with pancreatic tissue for a distance of from 2 to 7 cm.

The *vena porta* is formed in front of the processus uncinatus and the neck. The *superior mesenteric* vessels emerge beneath the neck and pass over the processus uncinatus.

The *body* of the pancreas is roughly triangular in shape and may be divided into the anterior, inferior, and posterior surfaces, separated from each other by the superior, anterior, and inferior borders. The anterior surface is covered by the peritoneum which enters into the formation of the posterior wall of the omental bursa, and in the greater part of its extent supports the postero-inferior portion of the stomach. The splenic vessels pursue a serpentine course along the superior border. The inferior surface looks downward and rests upon the coils of the small intestine and tranverse colon.

The digestive secretion of the gland finds its way into the intestinal canal by means of the *duct of Wirsung*. This duct begins at the tail, where it is formed by the union of a number of small ducts. It

increases progressively in size until it reaches the head, into the substance of which it dips, there coming into relationship with the ductus choledochus. Both ducts pierce the coats of the descending portion of the duodenum, terminating in its wall in a dilatation, the *ampulla of Vater*. Another duct emptying separately into the duodenum is the accessory *duct of Santorini*. It arises in the lower portion of the head and is much smaller than the one described above.

Physiological Considerations.— In order properly to understand the symptomatology of pancreatic disease, one must bear in mind that the organ has *two distinct types of secretion*, an *external*, which is concerned in the digestion of fats, and proteins, and an *internal*, the product of the islands of Langerhans, which is concerned chiefly in the metabolism of carbohydrates. Lesions of the pancreas may, therefore, be divided into two types, those affecting the islands of Langerhans, thereby interfering with the internal secretion, and those which involve the secreting parenchyma or the ducts, in this way either diminishing or preventing the entrance of the external secretion into the intestinal lumen. Only in extensive lesions, such as gangrenous pancreatitis, in which there has been a great destruction of gland substance, do we get symptoms suggestive of lessening or abolition of both types of secretion. This, however, occurs rarely, and then only in lesions which are both markedly advanced and very extensive.

Symptoms of Disturbed Pancreatic Function.— Lesions of the pancreas are productive of certain *symptoms* or signs which, in the absence of marked affections of the gastro-intestinal tract, are peculiar to, or strongly indicative of, the disordered function of this gland. Chief among these are the following:

1. Physical and chemical alteration of the fecal and urinary constituents.
2. Fat necrosis.
3. Hemorrhage.

Physical and Chemical Alteration of the Fecal and Urinary Constituents.— Since the pancreatic juice contains ferments which are essential for the proper digestion and absorption of the various food elements, it follows that affections of the secreting parenchyma or occlusion of the ducts that prevents the secretion from entering the duodenum properly, will cause a change in the character of the stool. Oser was the first to note the voluminous nature of the stools in pancreatic disease. Schmidt[2] and Pratt[3] have substantiated this observation. Pratt completely separated the pancreas from the duodenum in

dogs and observed that the weight of the dried feces was almost doubled though the animals received a uniform diet.

The fat in a true case of *steatorrhea* may be recognized by the naked eye, the stools appearing oily and butter-like. In other instances, they may be described as gray or asbestos-like. The appearance in this type of stool is no doubt due to the congealing and solidification of the fat and to the inadequate action of the pancreatic secretion upon the biliary pigments. Nothnagel has pointed out that the ability to digest fat varies normally and that steatorrhea is indicative of pancreatic disease only when certain diseases of the intestine or of its lymphatic apparatus have been eliminated. Such pathological conditions as amyloidosis, tabes mesenterica, tuberculous peritonitis, and those diseases which have as a clinical manifestation increased peristalsis would of necessity either hinder or prevent the proper digestion of fats.

Müller lays especial stress upon the split fat content of the stools. In health, according to his observations, the fecal fat which is not absorbed consists of 20 to 30 per cent of neutral fat and of 70 to 80 per cent of split fat. In pancreatic disease he has shown that the split fat content of the stool ranges between 22.4 per cent and 47.7 per cent. Katz[4] concludes that a diminution of this content below 70 per cent is strongly indicative of diminished pancreatic action. The reason for the inconstancy and even for failure of this symptom to appear is that, even in marked lesions of the pancreas, a sufficient quantity of the pancreatic ferments enter the duodenum properly to play their part in the digestion of the various food constituents.

Azotorrhea is less frequently observed, though as important a symptom as steatorrhea and often concomitant with it in the presence of undigested protein material in the stools of cases of pancreatic insufficiency. This symptom, first noted by Claude Bernard,[5] has been substantiated by Abelmann. The latter has been able to recover from 46 to 56 per cent of the nitrogenous ingestion from the feces of dogs in whom the pancreas has been extirpated. Schmidt lays emphasis upon the presence of undigested nuclear material in the feces, claiming that these elements can be digested only with the aid of the ferments present in the pancreatic secretion. He believes that the recognition of undigested nuclear material is strongly suggestive of diminished pancreatic efficiency. Fitz regards azotorrhea as clinically important only when gastric digestion is normal and when there has been no excessive ingestion of proteins. The presence of steatorrhea

and azotorrhea in combination is very strongly suggestive of disease of the pancreas.

Urinary Changes.— Cammidge[6] has endeavored to establish the presence of pancreatic disease by the detection of *abnormal urinary constituents*. He has applied the phenylhydrazin test for sugar to urine which has previously been treated by strong hydrochloric acid; when positive, a flocculent yellow precipitate results which, seen under the microscope, resembles a collection of fine hair-like crystals arranged in sheaf formation. This reaction, he believes, is strongly indicative of pancreatic disease.

In dogs with experimentally induced pancreatic lesions Hewlett[7] has observed the presence of a ferment capable of splitting ethyl butyrate into its component parts. Normally there is very little, if any, of this ferment present in the urine. He suggests this as a test for pancreatic disease accompanied by fat necrosis. Opie[8] also found a fat splitting ferment in a case of acute pancreatitis.

Fat Necrosis.— Balser[9] first directed attention to the fat necrosis condition. At autopsy, he observed the presence of small white opaque areas in the fat surrounding the pancreas. These nodules were subsequently shown to be composed of necrotic fat cells. Fitz[10] in 1889 suggested that the condition resulted from a pancreatic lesion. It is to Langerhans,[11] however, that we are indebted for our present knowledge of the subject. He was the first to associate the lesion with the peculiar physiology of the pancreas and was able to demonstrate that the fat molecule of the necrotic cell was split into a fatty acid and glycerin.

Flexner,[12] Opie[13] and others have demonstrated that when conditions favoring the escape of pancreatic secretion were produced in animals, fat necrosis of the tissue surrounding the gland resulted. The theory that the fat splitting enzyme is the active factor in the production of the necrosis is further borne out by the experimental work of Flexner[14] and of Wells.[15] The former was able to recover from the necrotic fat tissue an enzyme which split neutral butter fat with the liberation of free fatty acids. Wells was unable to produce fat necrosis after heating the watery extract of the pancreas.

Pathological Anatomy and Clinical Significance of Fat Necrosis.— The recognition of disseminated foci of fat necrosis is of great importance at the operation, this lesion usually being indicative of an acute hemorrhagic or gangrenous pancreatitis. More rarely, such con-

ditions as pancreatic calculi, gallstones, or carcinoma of the head of the pancreas will produce similar areas of necrosis.

On opening the abdomen, the omentum, mesentery, and fat surrounding the pancreas are found to be studded with rounded, sharply defined necrotic fat foci. These appear as whitish, or slightly yellowed, opaque areas, often surrounded by a slight zone of hemorrhagic infiltration. In the neighborhood of the pancreas there may be large confluent areas of necrosis. Less frequently the parietal peritoneum and diaphragm are also involved.

On microscopical examination the cell outline is usually found intact; the nucleus, however, is missing. At an early stage fatty acid crystals and later calcium salts of these crystals are contained within the cell boundaries. A few polymorphonuclear leukocytes also find their way into the necrotic tissue.

Pancreatitis.— The pancreas is subject to the same types of inflammatory lesions that affect other organs. Pathologically, therefore, two forms of inflammation may be recognized, the *catarrhal,* and the *parenchymatous.* The former, attacking the ducts, is not as frequently met with as the latter, which involves the cellular elements of the gland.

Catarrhal Pancreatitis.— Catarrhal inflammation of the pancreatic ducts is the outcome of extension of the infection along the duct of Wirsung. The duodenum, ampulla of Vater, and lower end of the common bile duct are the sources from which the causative bacterial agents are derived. The spread of the infective material is frequently facilitated by the presence of gallstones, which act as an impediment to the normal flow of bile. The condition is usually a concomitant of acute infective cholangitis, from which it cannot be clinically differentiated. As the process is infective in nature, operative interference is indicated. The removal of the impacted stone and drainage of the biliary passages is desirable.

Suppurative Catarrhal Pancreatitis.— Occasionally the catarrhal process goes on to suppuration. Usually, though, it accompanies suppurative cholangitis, or is the sequel of it. The same etiological factors are present in simple and suppurative catarrhal, pancreatitis, the spread of the inflammation by direct extension being the essential factor. The formation of pus in the latter case may be explained on the basis of the increased virulence of the infective agent, coupled with lowered bodily resistance.

The *symptoms* are those of suppurative cholangitis, being essentially septicemic and pyemic in nature.

The *prognosis* is grave; abscesses of the liver, of the gallbladder, or of the gland substance usually accompany this condition. Occasionally the inflammation becomes subacute in nature, ending in a single pancreatic abscess.

The *treatment* is surgical, pus cavities being evacuated and drained. Biliary calculi should be searched for and removed if possible. The bile ducts should be drained, in this way indirectly draining the pancreas.

The best *classification* of acute pancreatic inflammation has been submitted by Fitz,[10] as follows:

1. Acute hemorrhagic pancreatitis.
2. Gangrenous pancreatitis.
3. Suppurative pancreatitis.

As the result of his observations Fitz concluded that gangrenous pancreatitis usually, though not always, is the result of the hemorrhagic lesion. The same may be said of suppurative pancreatitis. The devitalized gland tissue becomes a suitable medium for the growth of the various organisms that secondarily invade it. This, of necessity, would result in suppuration and in the formation of abscesses.

Before the discussion of the various types of the disease, mention must be made of the conditions under which hemorrhages into the pancreatic tissue may occur. Hemorrhagic infiltration of the gland substance may be the result of traumatism or of malignant growths; petechial extravasations may accompany acute infectious diseases, such as purpura, or eclampsia. These hemorrhages, however, do not present anything peculiar to this organ.

Acute Hemorrhagic Pancreatitis (Hemorrhagic Necrosis of the Pancreas — Opie[13]).— This lesion should properly be called "hemorrhagic necrosis of the pancreas," as it has not the characteristics of a real inflammatory process. Experimental studies have shown that the primary lesion is a necrosis of the pancreatic parenchyma with subsequent erosion and necrosis of the blood vessel walls. The latter process is aided by the corrosive action of the pancreatic juice. The hemorrhage is the result of implication of the vessel walls. The inflammatory changes which may occur, Welch[16] believes, are due to the secondary invasion of the tissue by bacteria.

Experimental Hemorrhagic Necrosis of the Pancreas.— Opie, Flexner, and others have been able to reproduce this lesion in dogs by

injecting various irritants into the duct of Wirsung. Flexner[17] used different strengths of hydrochloric acid and artificial gastric juice. Opie[18] produced similar results by introducing bile into the pancreatic duct.

Hemorrhagic necrosis of the pancreas occurs more frequently in men than in women. Peiser[19] tabulated 121 cases, 79 of which occurred in men and 42 in women. In 41 instances of the hemorrhagic lesion Körte[20] found 37 in males and 4 in females; in 40 cases of the gangrenous type 21 were in males and 19 in females. Both the hemorrhagic and gangrenous varieties of this lesion occur between the ages of twenty and sixty.

Cholelithiasis.— Egdahl demonstrated the close relationship between cholelithiasis and acute pancreatitis. In 42 per cent of 105 cases the lesion was associated with gallstones. Opie and Bunting[21] have shown at autopsy that stones may lodge in the ampulla of Vater in such a way that the common bile duct and duct of Wirsung become a closed channel, thus preventing the escape of the biliary or the pancreatic secretions. Opie believes that at present no data are available to show the frequent coexistence of these two conditions.

Traumatic Necrosis.— A few instances of hemorrhagic pancreatitis have been observed following severe injuries to the upper abdomen. The pathogenesis of traumatic gangrene of the pancreas is obscure. Venous thrombosis may possibly explain the condition.

Of the variety of circumstances believed to favor the occurrence of pancreatic necrosis, the following may be mentioned: obesity, gastritis, duodenitis, and parturition. Undoubtedly, however, cholelithiasis plays the major etiological rôle.

Pathology.— The pancreas is much enlarged, is firm in consistence, and is covered by dark, cherry red blood clots. The gland itself presents a marbled appearance. This effect is due to the interposition between the hemorrhagic areas of normal parenchyma. The loose areolar tissue about the organ is infiltrated with blood. The lesser omental cavity is filled either with blood or with a blood tinged exudate. Scattered throughout the abdomen, though most frequently involving the retroperitoneal fat, mesentery and mesocolon are disseminated foci of fat necrosis.

The picture in the diseased areas is one of coagulation necrosis with destruction of epithelial cells, interstitial tissue and blood vessels.

Gangrenous Pancreatitis.— As this condition is usually the sequel of the acute hemorrhagic lesion, reference will be made here to its path-

ology. The symptomatological consideration of both types will be given later.

In cases of hemorrhagic necrosis which have survived for a week or more the gland appears blackish red or black, the tissue is dry, and is covered with altered blood. Still later, the organ becomes soft, black and friable. Foci of beginning suppuration may be seen at this stage.

Contained within the lesser peritoneal sac is a foul smelling, chocolate colored fluid, in which particles of necrotic tissue are seen to be floating. The foramen of Winslow is usually closed by adhesions, thus preventing communication with the general peritoneal cavity. The invasion of this fluid by organisms gives rise to an abscess formation, the walls of which are composed chiefly of necrotic fat tissue.

Erosion and perforation of the stomach, duodenum, or transverse colon may occur with the spread of the suppurative process. In such cases the passage by the bowel of shred or even of large sequestra of gland substance may be noted. The abscess cavity may pocket in the retroperitoneal tissue over the left kidney.

Symptoms.— The patient who may previously have been healthy is suddenly seized with violent abdominal *pain* localized chiefly in the epigastric region and slightly to the left of the median line. Fitz was able to elicit a history of *previous attack of indigestion* in the majority of his cases. Gessner[22] has shown that about 20 per cent of the cases were associated with symptoms of biliary colic, ofttimes accompanied by *jaundice*. The pain of onset is of great violence, and may be constant or may occur in paroxysms. It is increased by movement and accompanied very quickly by copious vomiting of bile stained material. The vomitus may later become dark brown or bloody.

Symptoms of shock now begin to manifest themselves. The patient looks drawn and ill; the respirations are shallow and rapid; the pulse rapid and of very poor quality. Cyanosis, especially marked about the face and abdominal wall, will often be observed, giving the patient a peculiar leaden hue. Halsted emphasizes this symptom. The symptoms of profound poisoning and of circulatory depression are probably due to the entrance into the blood stream of products of pancreatic autodigestion. Obstinate constipation occurs in the great majority of cases, and, in conjunction with the vomiting, may be mistaken for intestinal obstruction. There is rarely an absolute occlusion of the gut, this symptom being due in most instances to paralytic ileus.

The *abdomen* as a rule is not rigid, nor it is very tender, except on deep epigastric palpation. Later, when signs of generalized perito-

nitis begin to make their appearance, it becomes distended, tympanitic and rigid. Early epigastric distenton and tumor formation have been regarded by some observers as diagnostic. Opie, however, states that a tumor mass at this stage referable to the panereas is rarely if ever palpable.

Jaundice of varying degree occurred in 10 per cent of Gessner's cases.

The *temperature* during this stage varies. There may be a slight elevation. With symptoms of shock and collapse predominating, however, it is either normal or subnormal. *Leukocytosis* may be present during the first few days of the disease. This, however, is not constant.

Glycosuria is rare.

Symptoms During the Stage of Gangrene.— If the patient survives the acute attack, the disease becomes more chronic in nature. Clinically, it will be noted that the violence of the initial symptoms has begun to subside. The pain, though still constant, is diminished in intensity. It is confined to the epigastric region and is accompanied at intervals only by vomiting. A fever of slight or moderate degree replaces the normal or subnormal temperature which was present at the onset. Constipation, if present, disappears and may be followed by diarrhea.

Usually the presence of an ill defined tumor mass occupying the epigastrium and stretching over to the spleen is detected. This mass is separated from the liver by stomach tympany. It is due to the accumulation of fluid in the lesser peritoneal sac or to the presence of an hemorrhagic exudate about the panereas.

The presence of irregular fever, chills and leukocytosis gives evidence that bacterial invasion and suppuration of the necrotic tissue has occurred. Steatorrhea has rarely been observed, though chemical examination of the feces may show a diminution of the split fat content.

Glycosuria is an infrequent symptom.

Recognition of hemorrhagic necrosis of the panereas is dependent upon the mode of onset. The suddenness of the attack, the intense epigastric pain, vomiting, and the early incidence of shock are at once suggestive of this lesion. The absence of colicky pain, hyperperistalsis, and stercoraceous vomiting serve to eliminate intestinal obstruction.

Since this disease is often preceded by an attack of biliary colic, difficulty may be experienced in ruling out this condition. When an attack of biliary colic is of unusual severity, the pain having a tend-

ency to localize in the epigastrium, hemorrhagic pancreatitis is to be suspected, especially if symptoms of shock begin to manifest themselves. With perforating or phlegmonous cholecystitis the symptoms are confined to the right side beneath the costal margin.

When the disease has reached the subacute stage, the presence of an irregular epigastric tumor mass and the history of sudden onset with symptoms of collapse favor a diagnosis of gangrenous pancreatitis.

The *treatment* will necessarily vary with the condition of the patient. In the event of profound shock, surgical interference is dangerous. This, and other symptoms which are present, should then be combated by symptomatic measures. For collapse, hypodermoclysis, or infusion in conjunction with morphin, and external heat may be used. Warm saline enemata will relieve distention and also have a beneficial effect upon shock. Pain should be controlled by the use of opiates. Vomiting is best controlled by withholding food and by gastric lavage. Circulatory stimulants should also be administered.

The value of operation during the early stage, when symptoms of collapse and circulatory depression are predominant, has been much disputed. Mikulicz[23] maintains that early operation limits the extent of necrosis and that it diminishes the opportunity for the advent of such complications as venous thrombosis, and pyemia.

If, under treatment, shock diminishes, and the general condition of the patient improves, operation may be deferred until the process reaches a subacute stage. If the symptoms are not so influenced, or should the clinical picture show that the patient is failing, operative measures should be employed. Under nitrous oxid and oxygen narcosis, if possible, a median celiotomy should be performed. The lesser peritoneal cavity is entered through the gastrocolic ligament, and fluid or necrotic material, if present, is evacuated. Any free fluid should be rapidly mopped up or removed by suction. The pancreas may then be lightly incised, free drainage being afforded either through the operative wound or through a stab wound in the left loin. The bile ducts should now be examined to determine the presence of impacted stones. If the condition of the patient warrants, these should be removed and biliary drainage (p. 2119) instituted.

Acute Suppurative Pancreatitis.— The pathological lesion in this condition does not differ in appearance from a suppurative process occurring in other parts of the body. The organ is enlarged. It is soft, and as a result of this softening, one or more abscess cavities may form. Many of the reported cases of suppurative pancreatitis are really

peripancreatic abscesses, arising from a previous gangrene of the gland.

Suppurative pancreatitis is frequently secondary to acute hemorrhagic necrosis of the pancreas. It may also result from an ascending infection of the ducts. This is favored by the occlusion of the duct of Wirsung by gallstones lodged in the ampulla of Vater, pancreatic calculi, and pressure from without by neoplasms. Metastic abscesses also occur, but are rare.

Symptoms.— These are rather ill defined, as this condition is usually preceded by acute hemorrhagic pancreatitis, cholelithiasis, cancer, or ascending infection of the biliary ducts. In about 50 per cent of the cases, the onset is acute with intense epigastric pain, collapse, and vomiting. Many of these, no doubt, are cases of acute hemorrhagic necrosis with secondary infection and suppuration. At times, the onset is gradual with epigastric discomfort, and gastric symptoms of variable intensity.

Jaundice occurs in a considerable number of instances and may be the result of cholelithiasis or pressure upon the biliary ducts by a pancreatic abscess situated in the head of the gland (Thayer[24]).

Constipation is present in the majority of instances. Occasionally, an excess of fat and of undigested muscle fibers may be detected in the stools. At times the temperature is normal, though usually fever of a moderate or even high degree is present.

Abdominal palpation is rendered difficult by the presence of pain and epigastric distention. A tumor mass referable to the pancreas may, however, be detected in about one fourth of the cases.

Treatment.— Surgical efforts at relief should be prompt; delay is likely to be followed by such complications as general peritonitis or portal thrombosis. The abdominal incision and approach of the gland is similar to that described under the treatment of acute hemorrhagic pancreatitis. The abscess cavity is evacuated and drained in the usual manner. If necessary, additional drainage may be established by incising through either the right or the left costovertebral angle. Biliary or pancreatic calculi should be removed.

Chronic Pancreatitis.— This is doubtless the most common disease to which the pancreas is subject. So ill defined are its symptoms, however, that clinical recognition is difficult. Anatomically, two forms may be distinguished, namely, *chronic interlobular pancreatitis and chronic interacinar pancreatitis.*

In the former type the interlobular tissue is chiefly involved, the

gland being dense and hard, while its surface is either granular or nodular. Coarse bands of fibrous tissue separate the lobules of the parenchyma, causing pressure atrophy of the acini. The islands of Langerhans are normal.

Chronic interacinar pancreatitis is characterized by a diffuse fibrosis, which penetrates between the acini and soon involves the islands of Langerhans. The organ is firm in consistency and its surface is smooth. Proliferation of the interlobular tissue is slight and not constant. Interacinar pancreatitis does not occur as commonly as the interlobular form.

These cases occur more often in males and are usually accompanied by some form of biliary disease. Chronic interlobular pancreatitis follows occlusion of the duct of Wirsung by gallstones, pressure of neoplasms, etc. Ascending infection of the ducts, such as occurs in calculi, either biliary or pancreatic, associated with organisms of the colon group, is also a factor of great etiological importance.

In cases in which the etiology of the interacinar type is evident, the exciting cause seems to reach the organ by way of the blood vessels. Opie[13] and Lefas[25] have shown that this lesion is often associated with atrophic cirrhosis of the liver and arteriosclerosis. Alcoholism would, therefore, seem to play an important part in the production of this lesion.

Symptoms.— The clinical picture of chronic pancreatitis is very obscure, its symptoms usually being those of the conditions upon which it is dependent. At times, especially during periods of exacerbation, there is epigastric pain, nausea or vomiting, and, very often, jaundice. On deep palpation the head of the pancreas may be felt.

The temperature is usually normal, but fever may be present during an exacerbation of the disease.

The bowels are usually constipated and flatulence is present. In markedly advanced cases, the stools may show both steatorrhea and azotorrhea. Determination of the split fat content of the stool may establish the condition in certain instances.

The value of the Cammidge[6] reaction has been much disputed. Mayo Robson believes it to be an important aid to diagnosis, while Kemp[26] and others regard its value as dubious. Deaver[27] found it present in 25 per cent of his cases.

Treatment.— As chronic pancreatitis is usually an aberrant condition, arising frequently from some biliary disturbance, drainage of these ducts is indicated. In many cases this will cause a subsidence of

the inflammation. Drainage may be obtained by the performance of any of the following surgical procedures, cholecystotomy, choledochotomy or cholecystenterostomy. The last mentioned operation is indicated only in cases which are markedly advanced, as then it would be impossible to maintain external drainage for any great period of time. Biliary or pancreatic calculi, if present, should be removed.

Tuberculosis of the Pancreas.— Primary tuberculosis of the pancreas is rare. Mayo,[28] Aran,[29] and Chvostek[30] have, however, reported instances of its occurrence. The disease, as a rule, is associated with acute or chronic tuberculosis of other organs. The lesion rarely attains large proportions, is generally situated in the intralobular tissue, and rapidly undergoes caseation.

As the process does not tend to become widespread, symptoms of disturbed functional activity of the gland are usually absent.

Syphilis of the Pancreas.— The congenital lesion is characterized by an enlargement of the gland and by the proliferation of interlobular and interacinar connective tissue. In the acquired form irregular bands of scar-like tissue give the organ a rough, pitted appearance. Data referring to the symptomatology and diagnosis of this condition are not available.

Calculi of the Pancreas.— Pancreatic lithiasis is comparatively rare. In 1500 autopsies performed in Johns Hopkins Hospital, two instances were observed. Lazarus[31] has collected 57 cases from the literature.

The presence in pancreatic cysts of calculi suggests that their formation is favored by stagnation of the pancreatic secretion. Another etiological possibility is the temporary obstruction and ascending duct infection arising as the result of the temporary or permanent presence of gallstones in the ampulla of Vater.

Calculi of the pancreas are white in color, multiple, and, as a rule, lodge in the duct of Wirsung, causing dilatation of this channel. They are composed chiefly of the carbonate or phosphate of lime. As a result of their presence chronic interstitial inflammation of the gland occurs, which is at times attended with erosion of the duct.

The most frequent *symptom* is *pain* located in the epigastrium. It is either constant or colicky, and is likely to radiate to the left scapular region. In many instances, however, pancreatic and biliary colic cannot be differentiated. Associated symptoms are reflex circulatory depression and temporary collapse. The stools may show the presence of excess fat and calculi.

The *treatment* is similar to that of calculi in other locations. Moyni-

han[32] removed through the duodenum a calculus lodged in the duct of Wirsung. Straehlin and Roeber[33] incised the head of the gland, successfully removing stones lodged in this situation.

Pancreatic Cysts.— Cysts of the pancreas, though not of frequent occurrence, are of importance, since a consideration of them is necessary in the diagnosis of abdominal tumors. True pancreatic cysts arise as the result of partial or intermittent blocking of the pancreatic ducts or from spontaneous proliferation of the epithelium, and are to be distinguished from pseudocysts, which are dependent for their existence upon trauma or degenerative changes within the gland. The cyst wall in the former types has an epithelial lining, while in pseudocysts this is absent, the wall being formed of dense connective tissue only.

Pancreatic cysts are of the following varieties:

1. True cysts of the pancreas.
   a. Retention cysts.
   b. Proliferative cysts { cystadenomata. / epitheliomata cystica.
   c. Hydatid cysts.
   d. Congenital cystic disease.
2. Pseudocysts, usually traumatic in origin.

Retention Cysts.— Cysts arising as the result of obstruction of the larger pancreatic ducts by biliary calculi, pancreatic lithiasis or neoplasms are often small and accompanied by few, if any, clinical symptoms. A beaded dilatation of the pancreatic duct, described by Virchow[34] as *ranula pancreatica,* may exist, or numerous small cysts, filled with opaque, mucoid secretion, may be seen. The latter condition Klebs designated as *"acne pancreatica."* Solitary cysts of considerable size have also been observed. The walls of these cysts are formed of dense connective tissue, lined usually by a single, somewhat flattened layer of cylindrical epithelium.

Proliferative Cysts.— Proliferative cysts of the pancreas are usually multilocular and are characterized by a lining of columnar epithelium, which at times dips down into the cyst wall, and by the presence of papillary projections from the cyst lining. The contents are often blood stained. *Pathologically,* two types are recognized, the benign cystadenoma and the cystic epithelioma, which is malignant. In many instances the clinical course is the determining factor as to the malignancy of the growth, it being often difficult histologically to

differentiate between them. The malignant type is similar to that found in the breast (p. 1869).

Hydatid cysts and *congenital cysts* are exceedingly rare.

Traumatic Cysts.—Pseudocysts most frequently result from traumatism, although they may follow degenerative changes within the gland substance. They may be formed within the substance of the pancreas, or when large, they may cause rupture of the anterior surface of this organ, then being limited by the walls of the lesser peritoneal cavity. This point has been especially emphasized by Jordan Lloyd.[35] Körte in his investigation was able to elicit a history of previous trauma, either to the epigastric region or to the upper left abdominal quadrant, in about 28 per cent of the cases. In a number of instances pseudocysts, no doubt, follow acute hemorrhagic pancreatitis, the hemorrhagic and necrotic material undergoing solution and subsequent encapsulation.

The *fluid in pancreatic cysts and pseudocysts* is frequently mixed with fresh or altered blood. Occasionally it is clear and watery in appearance; more frequently it is viscid and more or less turbid. The specific gravity varies from 1.007 to 1.028 and the reaction is alkalin. More rarely it is neutral. Albumen is present. The presence of diastatic, lipolytic, or proteolytic ferments can be detected at times. Microscopic examination of the cystic contents may show red blood cells, epithelial cells, fatty acid crystals, necrotic tissue and at times cholesterin.

In the early stages the *symptoms* are often vague or absent; with the growth of the tumor, general symptoms begin to present themselves. Chief among these is epigastric pain, constant in type, and accompanied by distress after eating, by nausea, or by vomiting. The pain may, however, occur in colicky paroxysms. The symptoms of gastric distress, loss of appetite, vomiting, and consequent loss of weight result from pressure of the growth upon the stomach. Compression of the intestines and colon is usually accompanied by pain and constipation. Ascites or edema of the lower extremities may result from pressure either upon the portal vein or upon the inferior vena cava.

Functional disturbances of the gland are observed in only a few instances, and then, only when the cyst is accompanied by advanced pancreatitis.

In its growth the cyst, which at first lies behind the stomach, gradually displaces this viscus and comes into varying relationship with it

and the colon. This may be determined clinically by the inflation of these organs either by gas or by air. Most frequently the cyst grows directly forward, and, pushing the stomach upward, comes into relationship with the anterior abdominal wall between that organ and the colon. In these cases the tumor is covered by the gastrocolic ligament. In the smaller number of cases the cyst passes over the lesser curvature of the stomach, stretching the gastrohepatic ligament, and presents between the stomach and the liver. Occasionally cystic neoplasms arising from the head of the organ or springing from its tail penetrate between the layers of the mesocolon. The colon, in these instances, lies either below the cyst or crosses the summit of the mass.

Diagnosis.—The cyst occupies the upper abdomen, forming a semicircular bulging in the median line. It usually lies between the stomach and the colon, and, for this reason, these organs should always be inflated before arriving at a definite conclusion. These physical signs, coupled with a history of trauma to the epigastrium, or accompanied by an anamnesis suggestive of acute or chronic pancreatitis, should make one suspicious of the existence of a cystic neoplasm of the pancreas.

In *echinococcus cysts* of the liver, the tumor mass is more superficial in location and is not separated from the liver by an area of stomach tympany. A distended gallbladder is rarely covered by stomach or intestines, nor does it occupy the position of a pancreatic cyst.

Mesenteric cysts usually occupy the region of the umbilicus and are freely movable in all directions. Vaginal or rectal examination suffices to eliminate ovarian tumors.

Pancreatic cysts may be *treated* by incision and drainage, or by extirpation. Aspiration is a dangerous procedure.

A median celiotomy is made and the general abdominal cavity is protected by means of laparotomy pads. An aspirating needle or trocar and cannula is introduced and the bulk of the cystic contents is withdrawn. The opening into the cyst is now enlarged and the cavity carefully sponged out. Redundant cyst wall may be cut away. The edges of the opening in the cyst are then sutured to the parietal peritoneum and a drainage tube is introduced. The disadvantages of this operation are (1) that healing is slow, often being accompanied by the formation of a chronic pancreatic fistula; (2) the danger of recurrence; (3) the possibility of the formation of a postoperative hernia.

Extirpation was first successfully performed by Bozemann in 1882. Since the cyst is usually so markedly adherent to adjacent structures, the operation is beset with many difficulties and is dangerous also, because of the proximity of important blood vessels. The cyst is approached through a median celiotomy. The surgical procedure after that will depend upon existing conditions. The chief justification for total extirpation is the possibility of the cyst being of malignant nature.

Tumors of the Pancreas.— Carcinoma is the most common neoplasm of the pancreas, sarcoma, lymphosarcoma and adenoma occurring but rarely. The first may occur as a primary affection of the gland; more frequently, however, this organ is the seat of secondary carcinomatous invasion from the stomach, duodenum, or biliary passages. The tumor most frequently involves the head of the pancreas, causing compression of the pancreatic and biliary ducts. At times, the body or tail of the gland may be the site of disease. This, however, is an uncommon occurrence.

Carcinoma of the pancreas may spring from the glandular epithelium or may arise from the cells lining the secretory ducts. In the former instance it is of the spheroid type, and in the latter it is usually columnar. Spheroid celled carcinoma is the most common, the alveoli of cancer cells being imbedded in an abundant, dense, fibrous stroma. The tumor is then hard and of the scirrhous type. Less frequently the neoplasm is cellular and soft or encephaloid.

During the onset *symptoms* of more or less marked gastro-intestinal disturbance are usually present. The patient complains of loss of appetite, discomfort after eating, nausea, or vomiting. These symptoms which, at first, may be vague, gradually become more intense in nature and are accompanied by other manifestations of the disease. *Pain* is one of the earliest symptoms of pancreatic carcinoma. It is situated in the epigastric region, radiating toward the back and shoulders or sternum. It may increase continuously in intensity until death, or may occur in colicky paroxysms, which are suggestive of compression of the biliary or pancreatic ducts. The intensity of the pain, many observers believe, is due to pressure of the growth upon the celiac ganglia and nerves.

Icterus of varying degree is now usually present. The onset of the jaundice is insidious, gradually increasing in intensity until at last the skin attains a deep green black hue. The gallbladder is almost always dilated. This sign, which was first noted by Courvoisier,

may be regarded as pathognomonic of carcinoma of the head of the pancreas. Cases of jaundice due to obstruction by stone are usually accompanied by a contracted gallbladder.

With the further growth of the neoplasm a tumor mass may be detected. Mirallie[36] found that a tumor mass was palpable in about one fourth of 113 cases, which he collected. During this stage the patient is usually cachectic, great wasting and loss of strength having occurred, when markedly advanced.

Diagnosis.— The presence of epigastric pain, rapid emaciation, weakness, progressive jaundice accompanied by distention of the gallbladder and tumor are indicative of pancreatic carcinoma.

In cholelithiasis, the jaundice is not usually as severe as in carcinoma of the pancreas; the gallbladder is likely to be contracted, and with the history of repeated attacks of biliary colic, this condition may be eliminated.

With carcinoma of the liver this organ enlarges rapidly and jaundice, if present, is generally slight. Irregular nodules can be felt on the surface and on the edge of this organ.

Surgical effort in carcinoma of the pancreas is directed toward the removal of the neoplasmic growth, when possible; if not possible, then toward alleviation of the pain. Removal of the growth is difficult and is accompanied by a high mortality. *Partial pancreatectomy* is the operation of choice; the surgical technic is similar to that noted under pancreatic cysts. Villar[37] records 13 cases of partial pancreatectomy for malignant disease with 5 recoveries and 8 deaths.

For the relief of complications a variety of operations has been performed. When jaundice is of such severity as to cause much discomfort cholecystenterostomy or cholecystgastrostomy (Erdmann[38]) is indicated. Those cases which are accompanied by persistent vomiting and dilation of the stomach may be benefited by the performance of gastroenterostomy.

Wounds of the Pancreas.— These are of infrequent occurrence, this organ being well protected by other viscera and by its deep situation in the abdominal cavity. Injuries of the pancreas, when present, are the result of very severe trauma, always being accompanied by rupture of other viscera. Clinically, the symptoms are those of shock and internal hemorrhage, arising most frequently from rupture either of the liver or of the spleen.

Garré[39] has reported a case of transverse rupture of the pancreas

successfully treated by suture. Walther and Guillemin[40] record a similar case.

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PART XVII

THE SURGERY OF THE RECTUM AND THE ANUS

CHAPTER I

ANATOMICAL CONSIDERATIONS — EXAMINATION OF THE RECTUM

Anatomical Considerations.— The *rectum* was for a long time described as beginning at the lower end of the sigmoid flexure, i. e., from the level of the promontory of the sacrum to the levator ani muscle. This extends downward in the portio analis sive sphincterica. Recently, however, a new division has been made and the rectum is now regarded as beginning at the third sacral vertebra and extending to the anus. The portion of gut located above this is considered as part of the colon.

The rectum itself is divided into two parts, (1) the upper (*pars pelvica recti*), lying above the levator ani; (2) the lower reaching to and including the anus (*pars perinealis recti*). This division includes the ampulla of the rectum and its outlet. Thus the rectum itself is not covered by peritoneum, except where the pouch of Douglas comes in contact with it.

The *pars perinealis sphincterica analis recti* begins at the anus and extends upward and backward a distance of from 2.5 cm. to 3 cm. The two lower centimeters is surrounded by the external sphincter ani, the upper, by the internal sphincter, which is 3 cm. in width and is included in the upper 1 cm. of the external muscle. The lining of the anal canal is divided into three zones, the *cutaneous*, which extends to the lower edge of the internal sphincter; the *intermediate* (1.5 cm.), covered by mucosa with flat epithelium; and the *columnar*. In the last (zona columnaris) are located the sinuses of Morgagni, eight to ten in number, which represent small (size of a pea) blind sacculations of the mucosa. The protruding mucosa between these pouches forms a series of longitudinal columns. At the upper end of the zona columnaris the epithelium merges with the cylindrical cells lining the pelvic rectum.

The border of the perineal and pelvic rectum corresponds to the upper edge of the internal sphincter, where the levator ani muscle is attached. The pelvic rectum is 10 cm. to 12 cm. in length and, in

contradistinction to the anal canal, curves upward and forward, conforming to the hollow of the sacrum. In front, it lies in contact with the prostate, the vesicula seminalis, and the bladder.

The peritoneal fold — the *pouch of Douglas* — begins at the upper end of the prostate and, in the male, extends as low as the fifth sacral or first coccygeal verterbra; in the female it covers the upper end of the vagina. The pouch forms a fold in either side of the upper end of the pelvic rectum. The extraperitoneal portion of the rectum is inclosed in a special fascial sheath (*rectal fascia*). Between the latter and the muscularis of the rectum is a space which is filled with fat and contains the hypogastric vessels; these supply the rectum, the prostate, and the bladder.

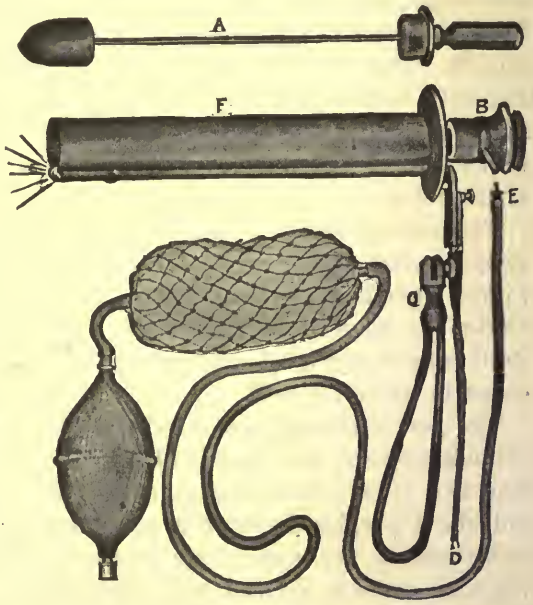

FIG. 945.— TUTTLE'S PNEUMATIC PROCTOSCOPE.

A, Obturator; *B*, plug with glass window closing end of tube; *C*, handle; *D*, cords connecting instrument with battery; *E*, inflating apparatus; *F*, main tube of proctoscope.

Examination of the Rectum. — Examination of the rectum has become a specialized art. However, there is no reason why its technic should not be readily mastered by the general surgeon.

There are four positions in which a patient may be examined for lesions of the rectum and anus. The first and most generally useful is the left lateral, so-called *Sims' position*. In the majority of instances, the position is sufficient for all examinations, the digital, ocular, by the speculum, or through the sigmoidoscope.

The *exaggerated lithotomy position* is ordinarily more convenient for operations and for examination of the rectum in corpulent persons.

The *knee chest posture* takes the weight of the abdominal viscera

from the rectum and is particularly serviceable in connection with instrumental examination of the upper rectum.

The *squatting posture* is useful in determining the extent of prolapse of the rectum, etc., during straining.

Digital examination is most important in determining the presence of spasm, the condition of the anal canal, the presence in the lower rectum of induration, ulceration, foreign bodies, polypi, stricture, neoplasms, hemorrhoids, etc., etc. The examination is limited to the lower four inches of the rectum. *Manual examination* (i. e., with the entire hand) has been abandoned.

Instrumental examination of the rectum is really a question of *light*. The form of speculum used is of minor importance. However, for the purpose the tubular speculum has replaced the older instruments. The ordinary tubular *proctoscope* or *sigmoidoscope*, into which the light is thrown by a head mirror, has been displaced by a similar apparatus, so fashioned as to allow of the introduction of an electric bulb which illuminates the field beyond the end of the instrument. The *dernier cri* in this connection is the *pneumatic proctoscope* of Tuttle[1] shown in Fig. 945.

The proctoscope is introduced through the anus with the obturator in position. As soon as the internal sphincter is passed, the obturator is withdrawn and the bayonet fitting plug, which contains a glass window, is inserted in the proximal end of the instrument. The plug being inserted in the tube, a very slight pressure upon the hand bulb will cause inflation of the rectal ampulla, so that the entire rectum may be visualized and the distal end of the instrument carried up to the promontory of the sacrum without coming in contact with the rectal wall. Further inflation permits introduction of the instrument into the sigmoid. The tubes vary from 4 to 14 inches in length. The *extent of the intestinal canal* visualized in this way has been increased to the *descending colon*. Narcosis is rarely necessary.

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CHAPTER II

MALFORMATIONS OF THE RECTUM

The division of these malformations into those originating in connection with the epiblast and those originating in the hypoblast and mesoblast, is made as follows:

Malformations of the Anus

1. Entire absence of anus.
2. Abnormal narrowing of the anus.
3. Partial occlusion of the anus.
4. Complete occlusion of the anus.
5. Anal opening at some abnormal point in the perineal, scrotal or sacral region.

Malformations of the Rectum

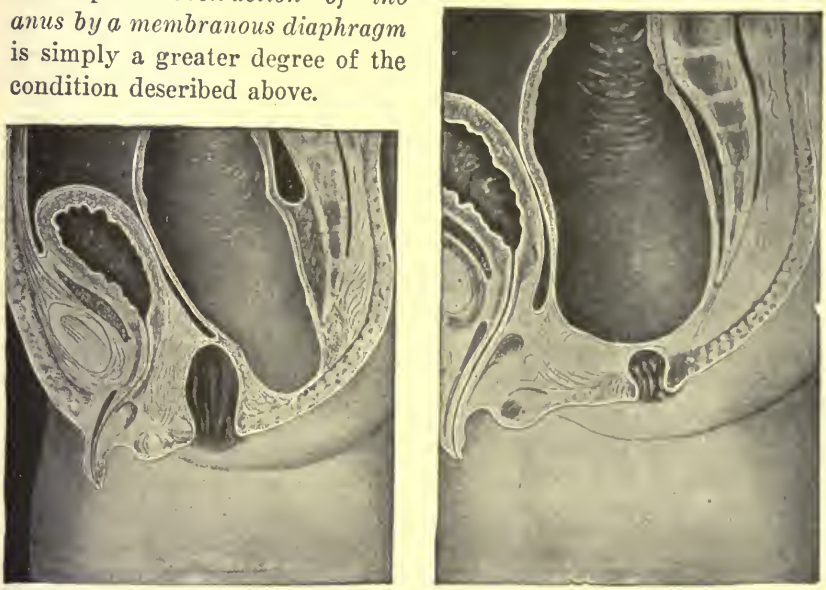
1. Rectum entirely absent.
2. Rectum arrested in its descent at a point more or less removed from the anus, the latter being normal.
3. Rectum opening into some other viscus, with the anus present in its normal position or absent.
4. Rectum and anus normal, with the exception that the ureter, bladder, vagina, urethra or uterus opens into it.

Malformations of the Anus.— In entire absence of the anus there is a depression of the skin or a corrugation at the point where the anus should be. In such cases the rectum may reach down almost to the skin, or may be arrested at a greater or less distance from it. However, the presence of a dimple does not indicate that the rectum is close to the perineum.

Abnormal narrowing of the anal canal may occur at any point in its course. The narrowing may be anular or very short, being formed by bands or membranes extending transversely or obliquely from side to side. The condition is likely to be attended with fissures which must not be confused with those due to hereditary lues.

Partial membranous occlusion of the anus consists in incomplete atresia of the perineal rectum by a membrane or fold of tissue. Sometimes it occurs in the form of a central raphé extending from the scrotum to the coccyx, with a small opening on one side or both. When the membranous occlusion is higher up, it it composed of mucocutaneous tissue, and is concentric or circular in form with a small opening in it; the latter varies in size.

Complete obstruction of the anus by a membranous diaphragm is simply a greater degree of the condition described above.



A *B*

FIG. 946.—*A*, CASE IN WHICH THE RECTUM DESCENDED POSTERIOR TO THE ANAL CANAL. *B*, RECTUM CLOSE TO ANUS.

An anal opening located at some abnormal point in the perineum or sacral region is ordinarily described as a malformation of the rectum itself and, in some instances, this is the case and a more or less developed anus is present. Examination of the adventitious opening usually reveals the presence of a more or less well developed sphincter muscle.

There is no fixed position in which the abnormal opening is found. It may be in the anterior or posterior part of the perineum, to one side or the other of the sacrum, or outside the gluteal fold.

It is important to differentiate these cases from those in which there

is an opening into the rectum that is remote from the natural one. If there is a sphincter at the abnormal opening, surgical measures of relief are of no avail, while in the other class of cases an effort in this direction is justifiable.

Malformations of the Rectum.— Entire absence of the rectum is recognized only by exploratory incision. No defects of the surface conformation are sufficient to predicate the entire absence of rectum. When a carefully conducted exploratory operation does not reveal the presence of a rectum, its absence may be assumed.

When the rectum is arrested in its descent and is more or less removed from the anus (the latter being normal), the malformation is ascribed to a failure of apposition of the enteron and the proctodeum, or the enteron may pass in a faulty direction and parallel the *cul de sac* of the proctodeum. The distance between the two varies widely. Sometimes it is only a few lines, while at others the blind terminal pouch of the rectum is found above the pelvic brim. In not a few instances, the pouch is divided into several compartments by various septa. When the rectum descends parallel with the anal canal and yet fails to establish apposition with it, the former is usually located posteriorly. The embryological deviations in this connection have been carefully studied by Keibel,[1] Hertwig,[2] and Laewen.[3]

In these cases the *diagnosis* is usually not made for several days after birth. The normal appearance of the anus does not suggest the true condition of affairs and the latter is not recognized until meteorism, vomiting, etc., are established. In some instances the finger in the anal canal detects an impulse transmitted from the blind pouch when the child cries.

Opening of the rectum into some other viscus — the anus being present in its normal position or absent — is by far the most frequent of all malformations of the rectum and anal canal (Leichtenstern[4]).

The various types of these malformations are designated in accord with the organ the rectum communicates with as follows:

1. Atresia ani vesicalis.
2. Atresia ani urethralis.
3. Atresia ani vaginalis.
4. Atresia ani uterini.

The *diagnosis* is, of course, easily established by noting the vicarious discharge of feces. The disturbances are due to infection and to the mechanical interference with the function of the organ into which the feces are discharged.

Not infrequently, the rectum and anus are normal, but have openings into other organs, such as the ureter, vagina, or uterus. Of these malformations, a rectovaginal communication is the most frequent.

Operative Treatment.— Operations for imperforate anus are comparatively modern. Tuttle[5] presents an interesting historical review in this connection.

Delay is not permissible. An effort should be made to determine the position of the rectum by inspection and by palpation. The use of the aspirator is dangerous. A straight incision is made from the point at which the anterior margin of the anus would be normally located, back through the skin and subcutaneous tissues, to the tip of the coccyx. If there is a rudimentary anus, the incision should begin at its posterior margin. The incision exposes the external sphincter or the fibrinous band which takes its place. These fibers are bluntly separated and the dissection is carried upward and backward into the hollow of the sacrum to avoid injury to the bladder or to other pelvic organs. When the rectum is found, an effort is made to loosen the pouch from its attachment and to deliver it through the incision; the pouch should not be opened at this time. If, however, this cannot be accomplished, the pouch is opened by means of the trocar and cannula and is emptied. The collapsed gut is then drawn out of the wound. When the closed pouch is delivered as far as the wound, it is opened with the knife. In either event, the mucosa of the rectum is turned out over the edge of the incision and sutured to the skin, thus keeping the raw surfaces from contact with the interior of the rectum. Most surgeons use interrupted silkworm gut sutures for the purpose.

When the rectum is arrested high up in the pelvis, the problem is often a difficult one. The operative methods employed for relief in this connection are numerous. Of these, the method of Vineent is described by Tuttle[5] in substance as follows: An incision is made from the base of the scrotum or the posterior wall of the vagina, and the coccyx and lower part of the sacrum are split in the center by means of heavy scissors. The edges of the wound are then retracted sufficiently to afford ample space for operative manipulations and for a view of the pelvic cavity. After the rectum is found and brought into its normal position, deep sutures are used to bring the bony and soft tissues together. Thus the pelvic frame is restored.

The *treatment of the anal cul de sac*, i. e., when the anus is fully developed and the rectum is more or less removed or has descended along the anal pouch, presents a difficult problem. The various

methods of establishing communication between the two have been widely discussed. The outcome of this would seem to be that the best results are obtained by dissecting out the mucosa of the anal pouch and bringing the rectal mucous membrane down to the margin of the skin and suturing it there.

Colostomy is made when it is impossible to locate the undescended rectum. Some surgeons employ celiotomy as a preliminary step, thus making it feasible to explore the pelvis from above and also to divert the fecal current from the pelvis while the perineal wound heals. A full discussion of the merits of the procedure is submitted by Tuttle[5].

In *abnormal narrowing of the anus* no operative measures are necessary in early life, provided there is sufficient space for egress of feces. As a rule dilatation with bougies accomplishes the desired end. If this fails, the narrowed portion is excised and the mucosa is brought to the anal margin, where it is sutured to the skin.

Partial occlusions by folds or band are treated by excision of the obstructing tissue. When partial occlusion is situated high in the anal canal, it is best to make radiating incisions into it and gradually dilate the canal.

Complete membranous occlusion is adequately treated by crucial division and subsequent bougie dilatation. The condition is rare.

The *treatment* of cases in which the rectum opens at an abnormal position in the skin, or of those in which communication exists with another viscus, is often a difficult problem and calls for the exercise of special methods for each case. These must per force depend upon the given problem and must be worked out on general surgical principles.

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CHAPTER III

FOREIGN BODIES IN THE RECTUM, INJURIES OF THE RECTUM, INFLAMMATIONS OF THE RECTUM

Foreign Bodies in the Rectum.— Foreign bodies gain access to the rectum from above. They are usually small (fish bones, chicken bones, seeds, kernels, etc.) and reach the rectum in from one to nine days after their introduction into the stomach. Foreign bodies rarely enter the rectum from below. However, some of astonishing size have been introduced by insane persons and by sexual perverts. Fecal stones deserve special mention. These either descend from the colon or are formed in the ampulla and may reach the size of the fetal head. As a rule, a fecal concretion is formed around a cherry or plum pit, or around other foreign substance, especially if the rectum is indolent, as happens in connection with diseases of the spinal cord. Proctitis and periproctitis often develop under these conditions.

Most foreign bodies are spontaneously ejected; others, especially those possessing sharp edges or projections, are caught in the mucosa of the ampulla and remain there. Bodies that are discharged with the fecal column often injure the mucosa and give rise to inflammatory conditions, fissures, and ulcerations; others burrow into the mucosa and are responsible for a *periproctal phlegmon* in which it is not unusual to find a fish bone, etc. Larger bodies have perforated the bladder, vagina, and even the peritoneum. The forcible introduction of foreign bodies gives rise to serious disturbances, such as obstipation ileus, catarrh, and evidences of irritation, including the discharge of mucus, pus, and blood.

Fecal concretions may remain in the rectum for a long time without producing serious disturbances. Ultimately, however, they are responsible for constipation alternating with diarrhea, obstruction and tenesmus. The last is the outcome of chronic irritation, the result of inflammation and pressure ulceration. If a digital examination is not made, these symptoms lead the surgeon far astray as regards the diagnosis.

The *treatment* consists of course in removing the offending agent. If the body is not expelled with an oil enema, it is best to narcotize the patient and remove the body through a metal speculum. If this fails, the sphincter must be divided. In some instances, when the body is located high up, celiotomy may have to be resorted to. The latter is not a difficult nor a particularly dangerous procedure.

Injuries of the Rectum.— The rectum is subject to the same injuries that occur in other parts of the body. They are most often produced by accidental force, although not a few are the result of unskillful efforts at therapeusis. The anterior wall of the rectum is not infrequently injured by the nozzle of a syringe. The latter has been made to penetrate into the perirectal tissue. Of the wounds in this situation, impalement injuries are of especial importance (Silbermark[1]).

These are divided into *intraperitoneal* and *extraperitoneal impalement injuries.* In many instances, the bladder or urethra, or both, are also injured. In the cases collected by Silbermark (224), the peritoneum was invaded in 89. Of the 89, the bladder or urethra, or both, were injured in three fourths of the cases; the sphincter was torn in 14, and in 26 cases intraperitoneal organs (omentum, ileum, colon, liver) were injured.

Projectiles also involve the bladder and peritoneum when the bullet enters from the front; lateral gunshot wounds are likely to splinter the bones and injure the vessels and nerves.

Obstetric injuries to the rectum are very common. They consist in lacerations or pressure necrosis. *Spontaneous rupture* of the rectum is rare, but occasionally results from increased abdominal pressure during great muscular exertion. The lesion has also occurred during straining at stool.

As regards *symptoms,* injuries of the rectum present two peculiarities, *first,* severe bleeding may take place above the sphincter and the blood may collect within the rectum, or, when the rectum is perforated, the bleeding may enter the peritoneal sac. In either event there may be no discharge of blood from the anus. The *second* peculiarity lies in the great danger of putrefactive infection. This may occur at the time of the injury or may result from *contamination* with feces (secondary infection). The latter is especially likely to occur when the sphincter is intact (absence of drainage). Under these conditions the blood and feces accumulated in the ampulla readily gain access to a wound in the mucosa of the rectum, and infection once developed in the periproctal tissue spreads rapidly in all directions.

The *treatment* of superficial wounds in the region of the anus does not present a special surgical problem.

Wounds produced by impalement, with injury to important organs, are likely to be followed by serious complications, and therefore, an injury of this sort demands systematic examination. Of course the character of the injury must be taken into account. The bladder is emptied by catheter, and the urine is examined. The patient is then narcotized, and the rectum thoroughly cleansed and examined. For the first twenty-four hours the evidences of peritonitis are to be watched for. In doubtful cases it is better to open the abdomen with the view of making primary repair than to wait for clearly defined symptoms in this connection.

Inflammation of the Anal Skin.— Intertrigo is an acute inflammation of the skin of the anal cleft. It usually occurs in corpulent persons as the result of friction, especially when the parts are not frequently cleansed.

The *symptoms* consist in burning, itching, and pain, which are sometimes unbearable, but soon subside when the patient is quiet.

The *treatment* during an acute attack consists in rest (in bed), and in cleanliness. A soothing ointment (zinc) may be applied. Recurrence may be prevented by indulgence in the refinements of the toilet applied to the region.

Pruritis ani is a mysterious affection. It is ascribed to sedentary habits of life, uterine diseases, catarrhal proctitis, polypi of the rectum, diabetes, etc., etc. (especially etcetera). Last, but not least, the nervous system is made responsible for the affliction.

The *symptomatology* is indicated by the name of the affection. The itching is at times almost unbearable, so that the patient not infrequently becomes melancholic.

The *treatment* is directed toward removal of the cause. Locally, carbolic acid and alcohol are useful palliative measures. Rave[2] reports startling results following the use of the X-ray. Tuttle[3] gives a long list of local applications.

Proctitis and Ulcer of the Rectum.— Simple acute catarrh of the rectum is caused by primary bacterial infection, or is the result of chemical irritation, especially the persistent use of cathartics. The effect of mechanical influences (foreign bodies) in this connection has already been alluded to. Of the latter, retained masses of feces play an important rôle. Proctitis is, however, most frequently secondary to inflammatory conditions of the small gut and colon or to some

definite lesion of the rectum, such as hemorrhoids, prolapse, polypi, carcinomata, etc. Proctitis is also often secondary (by extension) to infection of the prostate, the vesicula seminalis, and the female organs of generation (pyosalpinx).

Chronic Proctitis develops in sequence to the acute form or may be primarily such.

The *pathological anatomy* of acute proctitis does not present regional peculiarities (see Inflammations of Mucous Membranes, Part II, chap. x), except, perhaps, that ulceration is more common in this situation. The chronic form of proctitis is often attended with hyperplasia of the submucous connective tissue, and this is likely to cause the formation of areas of thickening (*proctitis polyposa*). Occasionally the inflammatory process invades the follicles of Lieberkühn and gives rise to hypertrophy of the lymphatic tubules, which undergo cystic degeneration (*mucous polypi*). A certain number of the lesions break down, forming ulcerations of greater or less dimensions, which are surrounded by the polypi. Finally, when a process of this sort heals, a stricture may be formed.

Acute Proctitis is expressed by the picture of inflammation. Pain is severe and often radiates to the sacrum, the groin, and the inner aspects of the thighs. Tenesmus is very distressing. The stool contains mucus and, at times, blood. In severe cases bladder disturbances occur and, not infrequently (especially in children), the mucosa prolapses (ectropium recti of Roser). Streptococcus proctitis is described by Simmonds.[4] The infection is likely to extend to the peritoneum.

Chronic proctitis often runs a protracted course, the only symptoms consisting in slight tenesmus and a feeling of weight in the rectum. This is usually attended with the ejection of pus or of mucus. After a variable period of time, constipation, alternating with diarrhea, develops. As a rule, the anal canal is fissured and the surrounding skin is more or less inflamed. In not a few instances the process extends to the periproctal tissues and an ischiorectal or a perirectal abscess develops. Ultimately, the symptoms of stricture are added to the picture.

The *treatment of acute proctitis* should be directed against its cause. The immediate symptoms are combated by placing the patient in bed with the pelvis elevated, by restriction of diet, and by frequent lavage of the rectum. Pain is best controlled by opium suppositories. The condition usually subsides in from eight to fourteen days.

Chronic proctitis presents a difficult therapeutic problem. In fresh

cases diet and lavage with astringent solutions, often relieve the situation. Of the medicinal agents, nitrate of silver (1:2000) maintains its position. Ulcers are treated with the silver stick and the actual cautery. Polypoid excrescences are best removed with the scissors. More advanced cases demand operative treatment. This is taken up in connection with stricture.

Specific Inflammations and Ulcers of the Rectum.— Specific inflammations and ulcers of the rectum are of various kinds.

Gonorrheal proctitis occurs most often in the female by extension (occasionally by rupture of an infected gland of Bartholin), or develops primarily in the passive pederast. The latter cause is most often operative in the female (Baer[5]).

The *clinical picture* is that of a mild proctitis, usually attended with ulceration. The condition is very likely to become chronic. The *diagnosis* is made by microscopical examination of the discharge.

The *treatment* consists of frequent lavage of the rectum with one of the silver salts (protargol, etc.).

Soft chancre occurs most often in the female. The lesion consists in multiple ulcers, or the process may take on a phagedenic form. Tuttle[3] shows some excellent illustrations of the condition.

Hard chancre is rarely found in the rectum. It may be located at the edge of the anus or in the anal canal. The ulcer is always solitary. The regional glands are usually inflamed.

Secondary lues involves the anus less frequently. It does so usually in the form of a nodular syphilid. However, the moisture of the region often causes the papillae to break down and become confluent, so that the anal outlet is eroded to a considerable extent. At this stage of the disease, the local lesion also appears in the form of condylomata, which extend to the labia and the scrotum, and may invade the rectum itself.

Gumma of the rectum occasionally occurs. The lesion may appear as a nodule in the lower segment of the rectum and in the anal canal, but is more likely to be diffuse in the form of *miliary gummata* which follow the course of the blood vessels (endarteritis, and phlebitis luetica). Gradually the various layers of the rectum, including the perirectal tissues, are infiltrated, so that finally little of the normal structures is left. As the process advances, the rectum is constantly subjected to acute inflammatory conditions more or less transient in character. As a rule, the affected area is covered by various forms of chronic inflammatory lesions, which make the diseased surface stand

out in marked contrast to the contiguous healthy mucosa. When healing takes place, the denuded tube presents a smooth, hard, connective tissue surface often arranged in grooves and elevations, not unlike the lingua geographica of tertiary lues (the elephantiasis recti of Rose). At this stage the luetic character of the condition is no longer discernible. Luetic disease of the rectum possesses characteristic localization. It begins, usually, close to the anus and extends slowly upward as far as the colon. The preponderance of rectal lues in women is explained on the ground that the primary sore is frequently situated at the posterior commissure, and that the infection is carried to the rectum by the vulvovaginal veins, which communicate with the rectal plexus. In the male the poison is carried from the penis to the inguinal region and into the iliac plexus.

Primary and secondary lues of the rectum is easily *recognized*. In the tertiary form the lesion may exist for a long time without attracting attention; ultimately the process presents the clinical picture of chronic proctitis with ulceration, and, finally, that of the formation of stricture.

The *treatment* is that of lues in general (p. 509).

Tuberculosis of the rectum is usually the outcome of infection from the intestinal contents. The bacilli are either swallowed with the sputum or introduced with food. Occasionally, no doubt, the infection is hematogenous in origin.

Tuberculosis infects the rectum through small erosions or gains access to the tissues through the lymph nodes, and is responsible for destruction of the mucosa, of the skin, or of the periproctal tissues. The cold abscesses

FIG. 947.— TUBERCULOUS STRICTURE AND ULCERATION OF THE SIGMOID.

developing in connection with the last event are discussed under periproctitis.

Superficial tuberculous infection of the rectum appears in three forms. The first, *lupus* of the anal skin, is rare and occurs in connection with healed fistulae. The second, the *verrucous or papillary form,* is also rare and appears in the skin and in the anal mucosa as true papillary proliferations, in which the microscope detects the tuberculous nodule. These may resemble luetic condylomata. The third takes the form of *ulcers,* which may appear primarily or in connection with abscesses and fistulae.

The *primary ulcerative form* occurs in tuberculous patients and has a tendency to extend around the lumen of the rectum in the form of a ring. It shows no disposition to heal. The patient soon dies of an exhausting diarrhea. In the rectum itself, tuberculous ulcers are comparatively rare (Quenu and Hartmann,[6] Tuttle,[3] Lartigau,[7] Sourdille[8]).

The *treatment,* aside from the general care of the patient, consists in cleanliness and in the extirpation of the infected tissues. The presentation of Tuttle[3] is especially complete.

Actinomycosis of the rectum is either *secondary* to a similar lesion elsewhere in the body or is *primary;* the latter is very rare. The lesion presents a clinical picture very similar to tuberculosis, from which it is differentiated by the microscope. See Melchior.[9]

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CHAPTER IV

FISSURA ANI — SPASM OF THE ANUS — PERIPROCTITIS — FISTULA IN ANO

Fissura Ani.— Fissura ani may be defined as a small, superficial ulcer situated in a cleft of the radiating folds of the anus, characterized by severe pain and spasm of the sphincter muscles.

Not all fissures are attended with the symptoms stated, and the latter may be present when there is no fissure. The fissure is usually *caused* by a slight injury of the anal mucosa (hard feces, foreign body). This is most likely to occur when the mucosa is thinned out by hemorrhoids (80 per cent, Quenu and Hartmann[1]). Ball[2] suggests that the primary erosion occurs in the valvulae Morgagni from pressure of particles of feces. In other instances the fissure develops in connection with eczema, erythema, injuries which result from pederasty, chancre, and gonorrhea.

The *symptoms* of fissure are characteristic. Defecation is exceedingly painful and is usually followed by slight bleeding. For some time after the bowel is emptied the patient experiences sphincteric spasm and a feeling of burning in the region of the anus. These disturbances are likely to be very distressing. In not a few instances the spasm extends to the urethral muscles and causes retention of urine. A spasmodic attack may be provoked by coughing, sneezing, by the expulsion of flatus, etc. In cases in which the symptoms of fissure are present without the existence of the local lesion, the condition is ascribed to nervousness and hysteria (Albu[3]). Occasionally the lesions heal spontaneously.

The *examination* of patients of this sort must be carefully and gently conducted. The *diagnosis* is not difficult except in the so-called hysterical cases. The great disproportion of the symptoms compared to the local lesion is due to neuritis of the terminal nerve fibers located in the fissure (Quenu and Hartmann[1]).

The *treatment* in light cases consists in keeping the stools soft and in the application of 10 per cent silver nitrate solution. The more

severe cases, which are not benefited by this method, are treated by *forcible stretching of the sphincter* under narcosis. The muscle must not be torn. The object is temporarily to paralyze it. As a rule, the fissure heals in four or five days. Should this fail, *partial* or *complete* division of the sphincter muscle with the knife is employed. The latter operation may be followed by incontinence of feces and is unnecessary.

Periproctitis.— Inflammatory and suppurative processes in the connective tissue surrounding the anus and the rectum are very common and are most often the result of infection following injuries.

The mucosa of the anal canal is frequently injured by fecal masses in their passage through it. This occurs most often in the lacunae Morgagni, especially when the latter form deep blind sacs. Most fistulae open into one of these sinuses. The infection gains access to tubular glands in this situation, which rapidly suppurate. The glands represent a deepening of the lacunae of Morgagni, called the sinuses of Hermann (Tavel,[4] Taler[5]). It is probable that infection may gain access to these glands without a solution of continuity of the mucosa, a condition of affairs similar to that presented in the crypts of the tonsil (Lexer[6]). Thus the infection readily gains access to the lymphatics of the periproctal tissue. This mode of invasion is particularly important in connection with tuberculosis. Periproctal suppuration is very likely to occur as a consequence of the various forms of proctitis, and more specifically is this the case when the latter process is ulcerative in character. Infection of the periproctal tissue also occurs by extension from contiguous parts, organs, and tissues, such as the female organs of generation, the prostate, and, occasionally, from the spinal column (gravity abscesses).

Suppurative processes in the periproctal connective tissue are *circumscribed* and *diffuse*.

Diffuse Periproctitis.— Diffuse suppuration is further divided into:

(1) The *diffuse septic form*, which occurs in connection with large wounds. This form, before the use of iodoform gauze tamponade, was frequently seen after extirpation of the rectum. The infection develops one or two days after the wound is inflicted and is usually putrefactive in character. The process advances with great rapidity, invades the retroperitoneal tissues, extends to the scrotum and penis and the trunk, and is fatal in from ten to fourteen days.

(2) The *diffuse periproctitis* is that caused by the *communis coli*.

This is attended with the *formation of gas,* and is less virulent in character than the one just described. However, a fatal outcome is the rule.

(3) The *diffuse gangrenous* appears to develop spontaneously (diabetes?). The affection is primarily gangrenous and involves large areas of the perineum, scrotum, etc. Occasionally, a case recovers after free incision (Abbe,[7] Wymann,[8] Gibbon[9]).

Circumscribed Periproctitis.— Circumscribed periproctitis is divided into *superficial* and *deep.*

Superficial abscesses develop beneath the skin (*subcutaneous*) and point near the anus. They may break into the ischiorectal fossa or into the submucous space of the portio analis. Submucous abscesses begin close to the anal canal. These may perforate through the skin, but usually break into the anus and are responsible for most fistulae. Occasionally an abscess of this sort extends into the ischiorectal fossa. Clinically, they follow the course either of a pyogenic infection or of tuberculosis. In the latter instance they give rise to little disturbance.

Ischiorectal Abscess.— Ischiorectal abscess is much less frequently seen than the forms just described. It is most often secondary to the submucous or subcutaneous forms of infection. Acute abscesses present rather a stormy picture of sepsis with severe local manifestations of inflammation. The deep location of the process may mask the *diagnosis* at first.

When these abscesses are not promptly opened they perforate in various directions, i. e., into the skin, the ischiorectal fossa of the opposite side, and the pelvirectal space, from which the infection may invade the retroperitoneal tissues. Death from sepsis is not uncommon.

Tuberculous ischiorectal abscesses pursue a chronic course. In not a few instances they are responsible for numerous fistulous tracts. An added mixed infection presents the picture of the acute process.

Pelvirectal abscesses are secondary to pathological processes involving the rectal mucosa (ulcers and strictures), or they are the result of perforation of contiguous abscesses. They occur most frequently in connection with pyogenic or tuberculous infection of the prostate. Their deep location makes the *diagnosis* difficult. Digital examination discovers an inflammatory thickening of the rectal mucosa and bulging of the rectal wall. Acute abscesses are attended with the general symptoms of the infection. These, like the chronic, extend in various directions and frequently riddle the periproctal tissues with pyogenic

sinuses, which open into the gut, into the ischiorectal fossa, or through the skin. Those arising from the prostate are most likely to rupture through the perineum or perforate the urethra.

The *treatment* consists in opening the abscess. *Subcutaneous and submucous abscesses* are opened by means of an incision radial to the sphincter, without dividing it. *Ischiorectal abscesses* are opened by an anteroposterior incision parallel to the sphincter. If the abscess has invaded the opposite side, the opening is best made through the posterior raphé, i. e., from the anus to the tip of the coccyx, and the two fossae are opened farther by carrying transverse incisions into them. *Pelvirectal* abscesses must not be opened through the rectum. As these usually arise from the prostate, a transverse incision is made in front of the anus and the abscess is entered between the urethra and the rectum.

Fistula in Ano.— Periproctal abscesses which open spontaneously rarely heal completely. The pyogenic cavity gradually fills with scar tissue, but leaves a tract lined with granulations. This means that all fistulae in this situation are sequential to periproctal abscesses. However, tuberculous fistulae deserve special mention. In a general way it may be said, that they almost always coexist with *pulmonary tuberculosis*. According to Melchior,[10] *diabetes* plays a causative rôle in a number of cases. Besides this, *lues* is responsible for fistula in ano in a not inconsiderable number of instances.

Tuttle[11] classifies fistulae as *complete* and *incomplete*.

Incomplete fistulae embrace all those cases in which the tract opens on one surface only. When the opening is outside the anorectal line, the term *blind external fistula* is used; when it opens within the rectum, it is called a *blind internal fistula*.

Complete fistula includes the cases having an external and an internal opening with a tract between them.

Fistulae are also classified, in accord with the tissues involved, into *subtegumental, submucocutaneous* or *submucous*. Those which pass outside the muscular apparatus of the rectum or anus are designated as *submuscular* or *subaponeurotic*.

In addition to this, fistulae are divided into *simple, complex*, and *complicated*. The *simple* fistula follows a direct course from one opening to the other; the *complex* has more than two openings, with more or less tortuous tracts between them, while a *complicated* fistula is associated with necrosis of bone, or communicates with a more or less remotely located organ, such as the urethra, the bladder, etc.

Finally, fistulae are divided on a pathological basis into *specific* and *non-specific*. The former occur in connection with tuberculosis, lues, and carcinoma, the latter with inflammatory lesions and trauma.

The *symptoms of fistulae* vary with the form of the lesion. The external incomplete cause the fewest disturbances — moisture, itching, intertrigo, slight discomfort during defecation, etc. The complicated, cause considerable disturbances; pus and feces are more or less constantly discharged, the skin of the anal region is irritated, defecation is painful, and tenesmus is likely to be distressing. Incomplete internal fistulae are characterized by indefinite pain in the rectum, which is accentuated during defecation. Pus and blood are often expelled with the stool. Intermittent attacks of proctitis occur.

FIG. 948.— FISTULA LAID OPEN OUTSIDE OF SPHINCTER, SO THAT THE LATTER CAN BE CUT SQUARELY ACROSS.

The *diagnosis*, when there is an external opening, is readily made with the probe. The presence of an internal opening is established by identifying the end of the instrument within the canal with the finger. As a rule, the inner opening is not very far from the anus. A blind internal fistula can only be recognized by examination with the proctoscope.

Complicated fistulae are recognized by the course they follow and by the history of the primary causative lesion (prostatic abscess, etc.).

The *surgical treatment* of fistula ani is based on the principle of *division of the soft part lying between the tract and the anal canal*, which converts the fistulous tract into a groove. Healing then occurs by granulation from the bottom of the wound. The typical operation is performed as follows:

After free catharsis and thorugh cleansing of the rectum and of the

superficial parts, a grooved director is passed from the external opening through the canal into the rectum (anal canal). The entrance of the end of the director into the rectum is recognized by the finger, and it is delivered out through the anus (Fig. 948). The soft parts, thus made tense, are divided with the knife or, still better, with the thermocautery. Bleeding is arrested, and the wound is tamponed with iodoform gauze. Postoperative pain is controlled with morphia. If the patient is properly prepared, the classic opium suppository may be omitted. On the fourth day castor oil is administered. After the bowel is emptied, the parts are cleansed and again tamponed. The latter is repeated after the daily stool until the wound is healed. Incomplete internal fistulae must be exposed with the speculum and divided from within the rectum in the manner stated.

Operations for the relief of fistulae must be considered from the *standpoint of the function of the sphincter muscles.*

In the ordinary subcutaneous and submucous fistulae (by far the most frequent), division of the soft parts does not damage the sphincters sufficiently to interfere with their function.

If the principle of converting the fistulous tract into a groove is to be carried out in ischiorectal and pelvirectal fistulae, the sphincters must be divided in part or completely. In this connection, the work of Matti[12] shows the following: When the external sphincter is divided, the function not only of the external, but also of the internal, is so impaired that incontinence of thin stool occurs. If the external is again sutured, the internal resumes its function. If the external is not entirely sectioned, the internal retains its function. Section of both sphincters is followed by complete incontinence. Isolated division of the internal sphincter is followed by gradual paralysis of the external.

This means that in operations for the relief of fistula, the external sphincter may be divided only to the lower (overlapping) edge of the internal. The problem comes under consideration in ischiorectal fistulae, the inner openings of which are located higher up than the internal sphincter. In these cases it is best to expose the tract by a parallel para-anal incision through which the inner opening is refreshed and repaired by suture. The external wound is treated as stated above. Primary division of the sphincters and their subsequent plastic repair is of doubtful utility (Tuttle[11]).

Multiple fistulae must all be widely opened and the primary opening in the rectum located — not always an easy task (Tuttle[11] submits

a free discussion of this class of cases; the work is freely illustrated).

Excision of the fistulous tract and primary suture (Fig. 949) consists in excising the fistulous tract, the course of which is indicated by

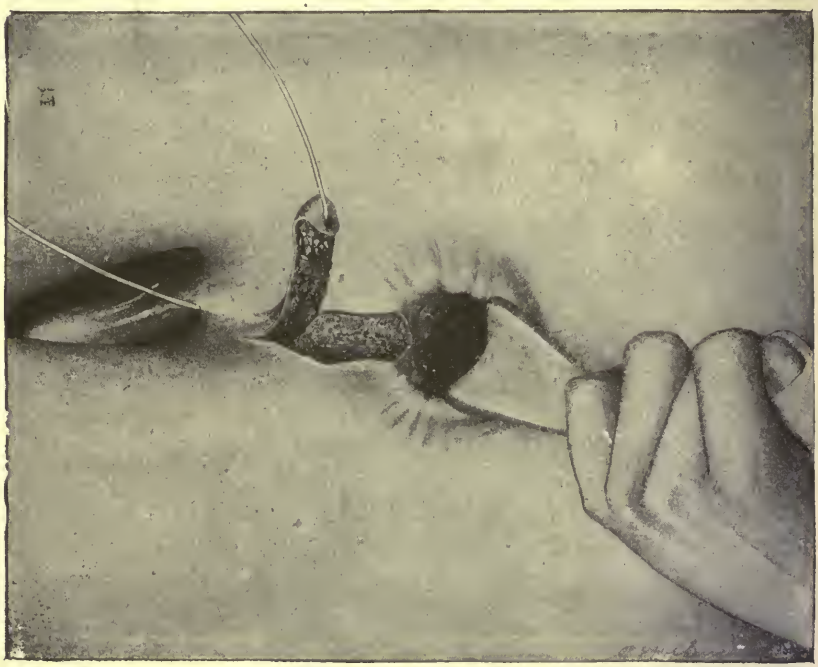

FIG. 949.— REMOVAL OF A FISTULA THREADED UPON A PROBE.

the probe, and in subsequent apposition of the soft parts by suture (Stephen Smith[13]). The method is frequently followed by healing.

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CHAPTER V

NARROWING OF THE RECTUM, STRICTURES, STRICTURE OF THE ANUS

Narrowing of the Rectum.— Narrowing of the lumen of the rectum may be restricted to its cutaneous portion (portio analis). This, however, is rare, and is the outcome of cicatrization in connection with ulcerative, phlegmonous processes and injuries, or it follows operative procedures such as the Whitehead hemorrhoidectomy, etc., in which too much tissue is removed. Congenital malformation responsible for narrowing of the canal is discussed above. The most severe narrowing of the anal canal is caused by scars, which at times constrict the aperture to an astonishing extent.

The *treatment* consists in (1) *gradual dilatation* of the stricture,

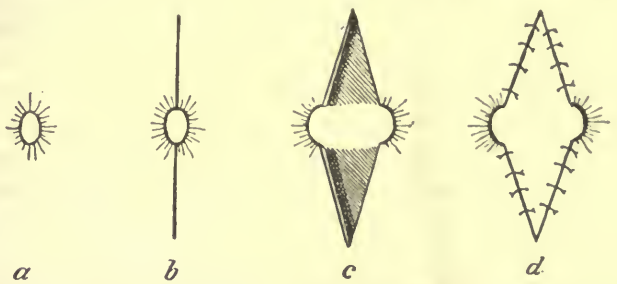

FIG. 950.— PROCTOPLASTY.

which is usually followed by recurrence; (2) operative methods, that based on the stomatoplasty of Dieffenbach being the most satisfactory.

The method is simple and is indicated in Fig. 950. The soft parts are split from the narrowed orifice (a) in front to the dam, and backward to the tip of the coccyx (b); the mucosa is separated in both areas and fastened to the skin, as shown in c and d. For additional methods see Rotter.[1]

Strictures of the Rectum.— Narrowing of the rectum is caused by:

1. *Changes taking place outside the rectum,* i. e., pelvic tumors.

Benign tumors interfere with defecation, but do not prevent it. *Malignant tumors* arising from the ovary and uterus may cause complete occlusion of the lumen. *Inflammatory* processes in contiguous organs, especially in those of the female genitals, cause pressure obstruction. In these cases the rectum is always displaced backward and compressed into the sacral hollow. The passage of a rectal tube is followed by the discharge of large quantities of feces. Occasionally, tuberculous peritonitis and perityphlitis (abscesses in the pouch of Douglass) compress the rectum.

2. *Changes in its wall,* which may be divided into those in which the *mucosa is healthy,* and *those in which it is involved in the pathological process.* To the first form belongs a characteristic process, described by Graser,[2] which originates in an inflammation of false diverticula in this situation. The diverticula occur in the pelvic colon and only in persons afflicted with cardiovascular disease. The latter is responsible for increased blood pressure, which in turn forces the veins through the muscularis. The pressure within the gut causes the mucosa to bulge out through the spaces in the muscularis thus created, in this way forming false diverticula. The latter are soon filled with masses of feces, which are responsible for ulceration, perforation, and suppurative inflammation in the subserosa which in turn is followed by the formation of scar tissue. This scar tissue shows a tendency to contract and, in doing so, forms a hard cicatricial ring (5 to 20 cm. in length) which finally obstructs the lower gut. As has already been stated, the mucosa takes a passive part in the production of the stricture, the active part being played by the deeper tissues.

The mucosa is, however, most frequently actively concerned in the production of stricture of the rectum. The most common lesion is that of carcinoma. In addition to this, stricture is caused by the healing of wounds and defects in the rectal wall.

Typical cicatricial stricturing of the rectum occurs in connection with *chronic inflammatory processes.* This type is represented by annular and cylindrical narrowing of the lumen of the rectum. The fact that lues is responsible for a number of lesions of this sort has caused the name *luetic stricture* to be applied to it. This, however, is misleading. Ruge[3] has shown that this form of stricture is not infrequently the result of healed tuberculous proctitis (Quenu and Hartmann,[4] Hartmann and Toupet,[5] Jeffries,[6] Panas and Valtat[7]).

The *seat* of the stricture varies, but is usually low in the rectum, just

above the anal canal. As a rule it is single, though occasionally more than one area of narrowing is present. A stricture is rarely more than 4 cm. in *length*. The lumen of the stricture may entirely occlude the rectum. The changes in the rectal wall at first vary with the character of the causative proctitis; later, a certain uniformity in the process is observed. The tube becomes rigid and increasingly hard; the mucosa, if any of it is left, is in a condition of chronic proliferative catarrh, with connective tissue infiltration. The mucosa above the stricture is usually more or less ulcerated, while that below is the seat of fissures, ulceration, papillary excrescences, fistulae, and abscesses. The submucosa is converted into a layer of dense fibrous tissue. Simultaneous perforation into contiguous organs (vagina, prostate, bladder) is not uncommon.

The *clinical picture* of stricture is always preceded by that of the causative proctitis (p. 2171). This may be present for many years. Persistent discharge of blood and pus, together with frequent defecation, should arouse suspicion of developing stricture. Later, obstipation dominates the picture. Recognizable alteration, i. e., deformation of the fecal column, is not so frequent as one would expect. The stool may be pencil shaped or ribbon shaped, or may resemble the feces of the sheep. If the stricture is high, the stool may be of normal conformation. Much importance may be attached to recurrent attacks of diarrhea alternating with obstipation, and to the presence in the stool of blood and pus. Defecation is usually attended with pain in the sacral region and with colic. The absorption of retained feces is attended with the picture of toxemia.

FIG. 951.—CREDÉ'S RECTAL BOUGIE.

The *diagnosis*, in strictures of the lower 9 cm. of the rectum, is readily made with the finger; those located higher are visualized with the proctoscope. The character of the process must be determined by the history of the case and by the serological and microscopical examinations.

The *medicinal treatment* of luetic strictures is efficacious only in the early stages of the disease; that of the concomitant catarrh of the other forms is similar to that of proctitis in general.

The *surgical treatment* is of two kinds, *mechanical* and *operative*.

The *mechanical treatment* consists in gradual dilatation of the stricture with the bougie. For the purpose, most surgeons use an instru-

ment made of metal or hard rubber. Of these, the bougie of Crède (Fig. 951) is perhaps most frequently employed. The method involves the danger of injury to the organ. Accidental perforation of the rectal wall is not exactly rare. It is avoided by introducing the bougie through the proctoscope. The bougie is allowed to remain *in situ* for from five to ten minutes. A number of ingenious instruments have been devised for the purpose of dilating the stricture (Tuttle[8]). *Rapid divulsion, electrolysis,* and *cauterization* are of doubtful utility, but are practiced by the specialist with varying success.

Partial proctotomy consists in incising the stricture in different parts of its circumference. The measure makes subsequent dilatation easier.

In *complete proctotomy* an incision is made in the posterior median line of the rectum, extending from the upper limit of the stricture down through the anus and tissues posterior to it, as far as the tip of the coccyx. A certain degree of incontinence follows the operation.

Colostomy, with the view of making it possible to carry out the rectal procedure with greatest safety, is a wise measure.

Extirpation of the strictured sector of the rectum is the operation of choice. This is rarely accomplished by the *transanal route.* The so-called vaginal route is more likely to succeed. The various methods of operating are taken up in connection with carcinoma of the rectum (p. 2207).

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CHAPTER VI

HEMORRHOIDS

General Considerations.— Hemorrhoids are dilated veins of the plexus hemorrhoidalis. The name hemorrhoid (a flow of blood) finds its origin in the most constant symptom — bleeding. The term *piles,* which means a swelling or tumor, is more correct. Why persons afflicted with piles should be made the subject of indecent wittieisms is not clear, but the fact remains that they are. Tuttle[1] tells us that Moses wished the affliction on "an impatient and rebellious people." Whether this was to be associated with trauma or not is not stated.

Hemorrhoids are classified as *external* and *internal.*

External hemorrhoids develop in connection with the inferior subcutaneous venous plexus, which communicates with the inferior hemorrhoidal vein, thus reaching the vena cava by way of the pudendal vein. The cutaneous plexus lies in the subcutaneous tissues of the anus.

Internal hemorrhoids lie in the internal zone of the superior hemorrhoidal plexus in the *submucosa of the lower ends of the columnae and the valvulae Morgagni* and extend upward from the lower edge of the internal sphincter muscle.

A hemorrhoidal nodule consists of dilated veins and capillaries (never arteries). The veins represent round, spindle shaped, or group-like dilatations and are often twisted. A number of these veins form a single nodule covered with connective tissue, which sends projections into the interior of the mass. As a rule, the nodule is the seat of thrombi and extravasations of blood. The investing mucosa is the seat of dilated capillaries and is usually in a condition of chronic inflammation. The protracted controversy as to the nature of the process seems to have been settled by the work of Silberberg,[2] who has shown that the lesions consist of dilated veins, the outcome of venous stasis and of inflammation.

The *causative venous stasis* is most largely influenced by intra-abdominal pressure, which is of course markedly increased by *obstipation.* The influence of the portal circulation is doubtful. On the

other hand, conditions which increase abdominal pressure, such as pregnancy, retroflexion and tumors, may be regarded as of causative moment. The condition is exceedingly common. Of 800 persons examined by Gunkel,[3] more than one half had hemorrhoids. Of these, 25 per cent presented symptoms. The lesion has been observed in children; Tuttle[1] reports two cases in infants (three years of age). The affliction is especially prevalent among the Jewish race ("goldene ader").

FIG. 952.— PROLAPSED INTERNAL HEMORRHOIDS.

The *symptoms* vary with the location of the hemorrhoids.

External Hemorrhoids. — External hemorrhoids appear at the anal outlet in the form of bluish nodules, varying in size from that of a pea to that of a hazel nut. These may be single, or multiple, or may form a complete wreath around the anus. The nodules enlarge when the patient strains and may be emptied by pressure. The superimposed skin is usually atrophic, but may be thickened from chronic inflammation. The dilatations cause only moderate disturbances, such as itching and burning. They rarely bleed. However, when inflammation is added (periphlebitis and thrombophlebitis), the attendant pain and discomfort are likely to be distressing. In this event, the nodules are tense, hard, and tender, and can no longer be emptied by pressure. Attacks of this sort last several days. Recession of the process leaves the nodule much shrunken (tabs). This is equivalent to spontaneous healing. Occasionally, a thrombolith develops.

Internal Hemorrhoids.— Internal hemorrhoids form (at the lower end of the columnae Morgagni) broad based or pedunculated prominences, which may be single, multiple, or form more than one ring around the circumference of the upper anal canal. The prominences

vary in size from that of a pea to that of a walnut. The tumors feel like soft folds of mucus and are covered by more or less inflamed bluish or vividly red (dilated capillaries) mucosa. They are freely movable upon the subjacent sphincter muscle. Occasionally the pulsation in the underlying submucous arteries is palpable. However, these are not dilated.

Internal hemorrhoids cause much more severe disturbances than the external, but may remain latent for a long time, giving rise to only a feeling of fullness in the rectum, and moderate pain after defecation. The first certain sign of the lesion is *bleeding*. The blood, which is usually expelled with the stool, coats the outer aspect of the fecal column and flows for a time after the bowel is emptied. The quantity of blood is usually small, but at times sufficient to produce anemia. The blood may emanate from a ruptured vein, but as a rule flows from ulcerated capillaries.

The second peculiarity of internal piles is their tendency to *prolapse*. The redundant mucosa is forced downward by the fecal column and finally emerges from the anus with the hemorrhoids. At first the extruded masses return spontaneously to the rectum; later, they must be manipulated back. In many instances the protrusion is grasped by the sphincters. When this happens, the mass may be *strangulated* or phlebitis may develop promptly. In this event, the prolapsed hemorrhoids appear in the form of a garland of red or blue, glistening nodules surrounded by a zone of inflamed, swollen mucosa. As the extrusion persists, it is attended with severe pain, tenesmus, retention of urine, fever, and, at times, with ileus. Gangrene is not uncommon. It is followed by ulceration, after which spontaneous healing occurs, or the area is left in a condition of chronic inflammation, within which fistulae and fissures are often found. Occasionally suppurative thrombophlebitis is responsible for general sepsis.

The third important symptom of internal hemorrhoids consists in *catarrhal proctitis*. This is evidenced by the expulsion of mucus and by the development of erythema and eczema at the anal region. The condition bore the name of *hémorroïdes blanches* (white piles) for many years.

Infection (phlebitis) gains access to hemorrhoids through erosions, fistulae, and fissures.

The *treatment* of hemorrhoids is *palliative*, and *radical*. (In all cases the underlying cause must be taken care of.)

Most hemorrhoids are benefited by *palliative measures*. Of these

the recently introduced hard rubber pessary shown in Fig. 953, is of therapeutic value. The instrument is introduced through the sphincters so that one bulb rests above them and the lower protrudes from the anus. The pressure caused by the shank is supposed to obliterate the delicate veins. The instrument is left *in situ* for hours at a time. The use of cold douches, massage, the application of ointments, etc., all have a tendency to lessen the symptoms attendant upon external hemorrhoids. Internal hemorrhoids may also be made bearable by the use of astringent injections or suppositories. Prolapsed hemorrhoids must be immediately reduced. For the purpose the patient is postured on the side and the pelvis is elevated. The extrusion is painted with 5 per cent cocain solution, is thoroughly anointed and gently manipulated back into the rectum. The pessary spoken of above may prevent extrusion of the masses between defecations.

Radical operative treatment is indicated (1) when repeated bleeding leads to anemia; (2) when internal piles show a persistent tendency to prolapse; (3) when inflammatory complications frequently occur.

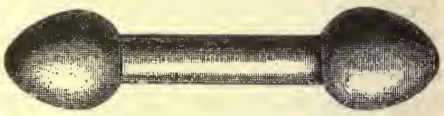

Fig. 953.—Hemorrhoid Pessary (Rotter).

Local anesthesia suffices in most instances.

Of the numerous methods of procedure that of the *clamp and cautery* still occupies first place. The patient is prepared as for the fistula operation and placed in the lithotomy position. Dilatation of the sphincters causes the hemorrhoids to prolapse. A single protrusion, or, if a wreath of hemorrhoids is present, the dome of a quadrant of the circumference, is picked up with a suitable forceps, and the pile clamp is made to grasp the base of the mass firmly in the direction of the long axis of the rectum (Fig. 954). The latter step is important; transverse application of the clamp is followed by stenosing cicatrization. The pile is then amputated with the scissors and the stump is cauterized. When the clamp is removed, the stump rarely bleeds. If, however, it does, the stump is again seized and deligated. Some surgeons reinforce the cauterized area with a running catgut suture. A strip of lubricated iodoform gauze is laid into the anal canal. Pain is controlled with morphia. Defecation is not permitted until the fourth day, at which time castor oil is given by mouth and an oil enema is injected. Complete healing requires about three weeks.

Whitehead's[4] operation, fully discussed and described by Tuttle,[1]

enjoyed a brief period of popularity. Most American surgeons have returned to the clamp and cautery.

Ligature of hemorrhoids (applicable only to internal hemorrhoids) is performed with *elastic* material or by *transfixion* and ligature to either side of the nodule. Neither of the methods possesses special advantages. On the other hand, there seems to be no serious objection to their employment.

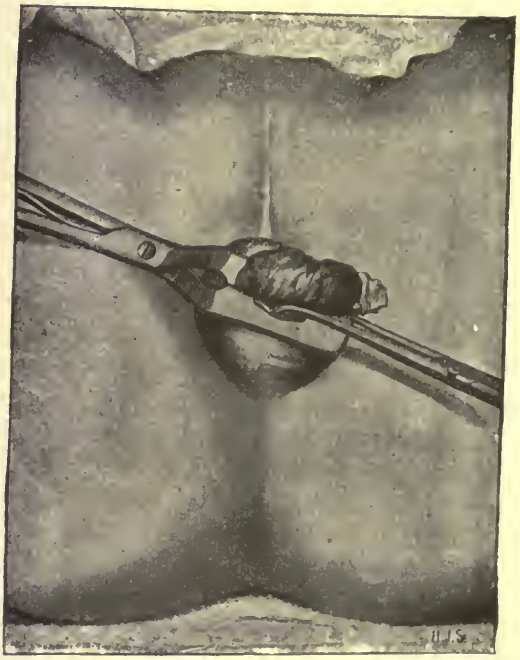

FIG. 954.—METHOD OF APPLYING THE CLAMP AFTER THE HEMORRHOID IS DRAGGED DOWN.

The *injection of 25 per cent carbolglycerin solution* into the pile leads to the formation of a thrombus in the dilated vein. At first, the pile is swollen and slightly painful. This, however, soon subsides. Gangrene and sloughing of the nodule are not rare. As a rule, the injection accomplishes shrinkage of only a part of the mass. The writer has seen two cases of fatal pylephlebitis follow the employment of the measure.

The term *high hemorrhoids* is applied to a varicose dilatation of the veins of the pelvic rectum (10 to 20 cm. above the anus). They appear as single or multiple varicose enlargements in the submucosa and bear some resemblance to polypi. The contiguous mucosa is usually in a state of chronic catarrh. Proctoscopic examination of patients afflicted with hemorrhoids reveals a lesion of this sort in 10 per cent of the cases.

High hemorrhoids give rise to severe bleeding. As they are not susceptible to digital palpation, their presence can be determined only through the proctoscope. The possibility of the existence of the lesion in cases of rectal hemorrhage must be borne in mind, especially in con-

nection with differential diagnosis. This argues for a complete examination of the lower bowel in all cases of bleeding from this part. The varicosities are easily destroyed with the cautery introduced through the proctoscope.

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CHAPTER VII

PROLAPSE OF THE RECTUM

General Considerations.— The term *prolapsus ani* is applied to protrusions of the anal rectum. This occurs very frequently in connection with piles. The extrusion consists of the anal mucosa. When a portion of the pelvic rectum is also prolapsed the term *prolapsus ani et recti* is used. In the latter event, the entire thickness of the rectum is protruded. The rectum is loosened in the periproctal tissue and is dissociated from its connection, i. e., from the levator muscle, prostate, vagina, coccyx, etc. When the intraperitoneal parts are prolapsed, the peritoneum is not separated from the gut, but is prolapsed with it. In this way the pouch of Douglas may emerge through the anus. In the latter contingency, the contents of the abdomen (small gut, sigmoid, ovaries, bladder) prolapse into the sac and a *rectal hernia* (*hedrocele*) is established. This is the sequence of events when the prolapse begins at the anal rectum and the parts above descend later.

In a considerable number of instances, the *upper portion of the pelvic rectum descends first* (Ludloff[1]). This is the outcome of the abdominal pressure on the pouch of Douglas, which is crowded into the hollow space of the ampulla and finally out through the anus. In this way the upper, subperitoneal portion of the rectum is invaginated into the lower. The finger detects the intussusception, and may be introduced into the fold between it and the intussucipiens. As the prolapse advances, the lower section of the rectum, including the anal mucosa, is extruded and this fold disappears. As long as the anus and lower segment of the rectum remain *in situ* the character of the prolapse may be recognized. After this the lesion presents no distinguishing anatomical characteristics.

The fully established prolapsus recti pelvini is shaped like a blunt bowling pin with a transverse lumen; the upper lip of the protrusion contains the pouch of Douglas. An *incomplete* prolapse refers to protrusion of the anterior wall of the rectum. When both walls are extruded the term *complete* prolapse is employed.

Normally the rectum is very snugly housed in the hollow of the sacrum and shows no tendency to wander from its secure environment. The invulnerable position of the normal rectum is secured primarily by the floor of the pelvis. The horizontal position of the perineum and the fact that the anus is located anterior to its lowest point constitute a condition of affairs antagonistic to the intra-abdominal pressure. This is materially aided by the sphincteric apparatus.

Under pathological conditions — in prolapse — the sphincters are relaxed, the levator ani is atrophied, and the anal canal is enlarged. When these conditions prevail, the posterior edge of the sphincter approaches the coccyx. The sphincteric prominence is flattened, the inner opening of the anal canal is displaced backward toward the lowest point in the pervis, and the course of the canal is no longer oblique, so that the dilated passage is subjected to the direct influence of the intra-abdominal pressure, and thus extrusion of the pelvic colon through the relaxed musculature is favored.

The rectum is intimately connected with its surroundings by connective tissue, which binds it in *front* to the prostate, the vesiculae seminales, and the bladder; on the *sides* to the parietal pelvic fascia; and *below* and *behind* to the levator ani, the coccyx, and the sacrum. When this tissue is relaxed, prolapse is favored.

Finally, the rectum is held upward by posterior prolongations of the pouch of Douglas (two peritoneal folds), which encase the upper sector of the organ and connect it with the sacral promontory. When the pouch is abnormally long, extrusion of the rectum is favored.

Etiology.— From this it is apparent that the predisposing causative factor in the production of prolapsus recti consists in weakening of the postanal rectal floor (preëminently the sphincter muscles), to which may be added, loosening of the attachments of the rectum from its surroundings, relaxation of the rectal wall, and an abnormally dependent fold of Douglas (Rotter[2]).

The faulty position of the anal canal and the abnormal depth of the pouch of Douglas may be *congenital;* to these causative factors may be added an unusually long mesorectum and an abnormal relaxation of the musculature in congenitally feeble persons.

Acquired predisposing factors include a number of conditions. Of these, chronic catarrhal proctitis has an important influence. Inflammatory processes of this sort are not limited to the mucosa, but also involve the submucosa, the intermuscular and the periproctal connective tissue, and, when they persist for a protracted period of time, are re-

sponsible for their relaxation. Overactive peristalsis weakens the rectal musculature and produces insufficiency of the sphincters, which ultimately exist only as a gaping ring. The pelvic rectum is then readily forced downward. This is especially frequent in children suffering from prolonged diarrhea, and in multiparae. It is not infrequently sequential to wasting diseases. In addition to this, mechanical stretching of the sphincters, such as occurs in passive pederasty, etc., and paralysis of these muscles concomitant with lesions of the nervous system, favor extrusion of the rectum.

The conditions stated, all of which loosen the attachments of the rectum and weaken its tone, favor the prolapse, but the extrusion itself is the result of increase in abdominal pressure coincident with each defecation. The oftener the latter occurs, the more is its influence operative. Thus chronic constipation becomes the most important causative factor in prolapsus recti, especially in women in whom the pelvic outlet is weakened, and in children with chronic diarrhea.

Symptoms.— The onset of the lesion is heralded by the protrusion of small sectors of the mucosa during defecation; this usually occurs at either or both sides of the anus. Later, larger portions of the rectum protrude. Prolapse of the rectum follows that of the anus. In other instances, the rectum prolapses first, and the anal canal is everted later. At first, the protrusion is spontaneously reduced. When the sphincter is still operative, it is likely to grasp the protrusion and strangle it. This gives rise to venous stasis, swelling, and bloody transudation, so that reposition is difficult to accomplish. Repeated prolapse is ultimately responsible for almost constant protrusion of the mass.

The size of the prolapse of course varies very widely, i. e., only the anal mucosa may protrude or the entire rectum and the sigmoid may be extruded. The protrusion forms a sausage shaped tumor, the walls of which are thickened. The opening of the gut is located at its lower end. The mucous membrane covering the tumor is usually in a state of chronic irritation. Occasionally incarceration of the protrusion leads to gangrene and fatal infection. If the prolapse remains extruded for a long time, the mucosa becomes dry, covered with crusts, and leathery in appearance. Incontinence is the rule.

In very large protrusions, the gut occupies the prolapsed pouch of Douglas. This enlarges the protrusion, sometimes to an enormous extent (size of the fetal head). The mesenteric attachment of the gut causes it to lie in the posterior portion of the prolapse. If the gut be-

comes adherent to the peritoneal sac, the prolapse becomes irreducible.

Treatment.— The treatment of rectal prolapse must be based on its pathogenesis and etiology.

The methods of treatment are divided into the *non-operative* and the *operative.*

Non-operative methods are chiefly employed in children. The measures of relief are directed toward correction of the cause of the prolapse, i. e., the gastro-intestinal disturbances. A discussion of this aspect of the situation does not belong here. The child must not be allowed to defecate while in the sitting position. Strange to say, the squatting position is attended with less increase of pressure than the former. At first, the child defecates while in the horizontal position (surely the acme of luxury). The nates should be held in apposition with strips of plaster; these are changed every few days. It is important that the prolapse be immediately reduced.

If removal of the cause and the methods stated do not accomplish the purpose, massage and mechanotherapy may be given a trial, The results in this connection are astonishing and yet, after all, they are based on the etiology of the condition. The use of retention apparatus is not satisfactory.

The *operative treatment* is divided into that of the anus and that of the pelvic rectum.

Prolapse of the anus is best treated by cauterization of the extruded mucosa of the anus and lower rectum. The measure is carried out as is described in connection with hemorrhoids, except that a much longer fold of redundant mucosa is picked up with the clamp. After cauterization the edges of the burned areas are approximated with a running catgut suture, which also constricts the subjacent blood vessels (Rotter[2]). The Whitehead operation, alluded to in connection with internal piles, is also used in this connection by British surgeons.

In the light of the pathogenesis of *prolapsus recti pelvici,* operative measures may be directed toward (1) the weakened pelvic floor; (2) the relaxed periproctal attachments of the rectum; and (3) the dislodged pouch of Douglas.

Considering the dominant influence of the muscular pelvic floor in the development of prolapse, measures involving correction of the fallacy in this connection deserve preëminence. Of these the simplest is that of Thiersch.[3] The method consists in encircling the anus with a subcutaneous silver wire suture. The needle enters at the posterior

raphé and comes in and out of the skin at various points, and is reintroduced until it emerges close to the point of insertion. The wire is tightened and twisted and finally buried. The method is successful in a certain number of early cases of moderate degree.

Excision of a V-shaped sector of the posterior aspect of the sphincters, and subsequent suture, is followed by incontinence of feces. Resection of a sector of the mucosa at the posterior quadrant of the prolapsed rectum, and approximation of the sphincter muscles by buried sutures, gives better results. Duret[4] removes a sector of mucosa from both the anterior and posterior surfaces of the prolapsed mucosa and, after refreshing the skin, sutures the denuded surfaces together. The operation is fully described by Tuttle.[5]

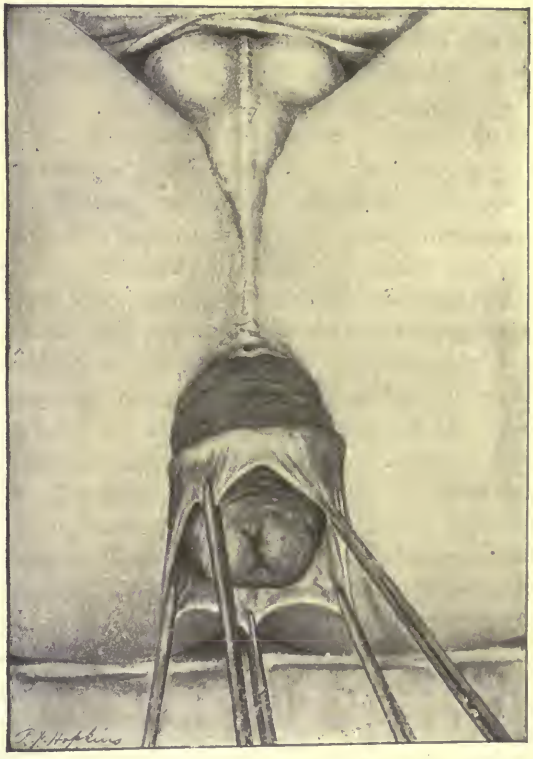

Fig. 955.—Delorme's Operation for Procidentia Recti.

A number of modifications of the various methods designed to meet the indications find culmination in the operation of Delorme,[6] which is performed as follows: After forcible dilatation of the sphincters, the prolapse is completely delivered and the mucosa seized with the artery forceps close to the anus, and the prolapse denuded as shown in Fig. 955. Thus a cylinder of mucosa may be excised, 10 to 15 cm. in length.

Now the extruded rectum consists of denuded muscularis from the anal margin to the upper edge of the prolapse. A series of reef knot sutures is placed, passing in and out of the muscularis in a longi-

tudinal direction, so that when they are tied, the rectum is thrown into a number of transverse folds, which form a thick protrusion. This is readily reduced and brought within the anus. If necessary, the anus may be narrowed by means of a few deep sutures placed in the anococcygeal area. Finally, the mucous membrane cylinder is amputated close to the anus, and the stump of mucosa sutured to the anal skin. The ultimate result is good.

The second group of operations — the *suspension methods* — is designed to reëstablish the attachments of the rectum within the pelvis by fixing the organ within the small pelvis — *rectopexy* — or the colon higher up — colopexy.

Rectopexy, as modified by Fowler,[7] is described by Tuttle,[5] as follows:

"The prolapse is dragged down to its full extent and held forward by an assistant. A curved incision about 2 inches in length is made midway between the coccyx and the anus. This is carried through all the tissues into the retrorectal space. With the fingers or a dull instrument, introduced through this incision, the rectum is separated from the coccyx and sacrum posteriorly as high up as the attachment of the mesorectum and on the sides as far as the attachment of the lateral ligaments. The anterior surface of the bone is then gently curetted to remove all the fatty tissue and freshen it. At this point the assistant reduces the prolapse, and, with his fingers inside the gut, inverts and brings it out through the incision, while the operator catches the protrusion and drags the gut down as far as it will come, usually a little less than the amount prolapsed through the anus. The external surface or muscular wall of the gut thus exposed is then curetted, as was the sacrum. Silkworm gut or silver sutures are then passed transversely through the muscular layer, embracing as much of the circumference of the gut as is possible; they are placed ½ inch apart, and the ends left 6 to 8 inches long. After the sutures have been placed, the ends of the upper ones are each in turn threaded on a long, curved needle and carried up through the wound to the highest point of the separation between the rectum and the sacrum, where they are made to penetrate the tissues, and are brought out through the skin on opposite sides of the bone. The other sutures are treated in like manner, each being brought out ½ inch lower than the preceding one. The ends are then drawn taut, and the prolapse is thus dragged up into the hollow of the sacrum, where it belongs. A pad of gauze is laid over the sacrum, and the sutures tied over this to avoid their cutting into the skin. The gut is thus anchored in close apposition to the sacrum, to which it

unites in due time. The external wound is closed by buried catgut and subcutaneous sutures. If the sphincters are much relaxed or overstretched, a ligature of kangaroo tendon is passed around the anus at the upper margin of the external sphincter and tied over the index finger introduced through the anus. This narrows the anal outlet and contracts the muscle, thus contributing to the cure. The bowels are confined for eight days, when they are moved by enemata. The patient is required to remain in bed and use the bedpan for three weeks, after which time he may be allowed to go to the toilet. The anchoring sutures are left in from ten to fourteen days."

Tuttle's description is illuminated by a series of excellent illustrations.

Colopexy, or ventral fixation of the sigmoid, is a simple procedure. The abdomen is opened through an oblique incision in the left hypogastrium; the sigmoid is drawn upward and fastened to the wound as for fecal fistula (v. Eiselsberg,[8] Pachnio[9]).

The third method of operating attempts to correct the prolapse by *plication of the pouch of Douglas.* The abdomen is opened through the median line; the sigmoid and rectum are drawn upward as far as is possible and the pouch of Douglas obliterated by a series of transverse continuous sutures placed between the posterior wall of the uterus and the anterior wall of the sigmoid. The operation is supplemented by repair of the perineum.

The last, and least often practiced, method of operating consists in *resection of the prolapsed rectum;* the prolapse is amputated and the ends are united by circular suture. The method most frequently employed is that of Mikulicz.[10] The procedure is as follows:

The patient is placed in the lithotomy position with the hips well elevated, and the prolapse dragged down as far as is possible by traction forceps. It is then clamped by two volsella forceps and held in this position by assistants. The elevation of the hips allows any coils of small intestine to slip out of the peritoneal pouch, and thus avoids the danger of wounding them. An incision is made through the mucous membrane upon the anterior surface of the gut at the margin of the anus. Dissection is carefully carried through the entire thickness of the intestine, all bleeding being checked as it occurs, until the peritoneal cavity is opened. This brings into view the intussuscepted portion of the gut. This membrane should be cut through and its upper edge should be sutured to the peritoneal edge of the wound in the anterior layer of the prolapse. Thus, step by step, the peritoneal

pouch is closed. The entire thickness of the intussuscepted gut is now cut through, little by little, and its muscular and mucous layers sutured by interupted silk or chromicized catgut to the mucous membrane surrounding the margin of the anus at the site of the original incision. In this manner the entire prolapse is excised, and end to end union of the gut is accomplished. The ends of the sutures in the muscular and mucous layers should be left long in order to steady the parts and prevent their retraction while the operation upon the other portion of the circumference is being made. All bleeding points should be caught and twisted or ligated during the operation. After completing the excision, if the edges of the mucous membrane are not in accurate apposition, a fine running suture of catgut should be applied around the entire circumference to accomplish this. The long ends of the sutures are then cut off, the wound is dusted with iodoform or boric acid, and over this several layers of flexible collodion are applied.

With regard to *selecting the method of operation,* Rotter[2] says in substance: In *prolapse of the anus* and a small sector of pelvic rectum, the clamp and cautery method is uniformly satisfactory.

Of the methods of procedure used for relief of *prolapsed pelvic rectum, rectopexy and colopexy* have not accomplished the purpose; *plication of the pouch of Douglas* is followed by relief, provided the perineum is repaired. On the other hand, *measures which reconstruct the floor of the pelvis* are followed by relief in most instances; of these, the *method of Delorme* is as useful as any in cases in which the prolapse is reducible. The *v. Mikulicz method* should be reserved for irreducible prolapse.

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CHAPTER VIII

TUMORS OF THE ANUS AND RECTUM

Tumors of the Anus.— Benign tumors of the anus are comparatively rare. Of these, condylomata are the most common. They are frequently due to chronic irritation, such as gonorrhea in women.

The *treatment* consists in excision of the growths and the application of cautery to the bleeding bases.

In addition to this, *papillomata, atheromata, hemorrhagic cysts,* and small *fibromata* occasionally appear in this situation.

Carcinomata in the region of the anus are rare in comparison with those involving the rectum itself. The process seems to develop at the site of old fissures, fistulae, and piles.

The growth begins on the skin or in the mucosa and is either superficial or deep, i. e., the disease emanates from the epithelium of the surface or of the hair follicles, from sweat glands or from sebaceous glands. The process does not present any regional peculiarities, except, perhaps, that it shows a tendency to form cauliflower-like excrescences. Occasionally, a carcinoma of the anal canal appears primarily in the form of a nodule which later breaks down. It is likely to be mistaken for sarcoma.

The process usually follows a stormy *course.* The inguinal and pelvic glands are invaded early, so that the disease soon becomes inoperable. Pain is likely to be severe.

Operative measures of relief must be employed early and must be radical. No attempt should be made to save the sphincters. See carcinoma of the rectum.

New Growths of the Rectum.— As elsewhere in the body, tumors of the rectum are divided into those originating in the connective tissue, and those arising in connection with the epithelial elements.

Of the benign *connective tissue growths, lipomata, fibromata,* and *myomata* are almost surgical curiosities. *Angiomata* develop occasionally in the submucous tissue; they are likely to be attended with fatal bleeding.

Of the *malignant connective tissue growths,* spindle cell *sarcomata* are the most frequent and are likely to be of the melanotic variety. Quenu[1] has collected 14 cases of this sort. The outlook in this class of cases is no more favorable than it is in other portions of the body. However, more or less prolonged freedom from recurrence after extirpation of the disease has been reported by Paul,[2] Ball,[3] Von Esmarch,[4] Tuttle,[5] and others. Tuttle had two cases; one did not show recurrence at the end of five years.

Echinococcus and dermoid cysts are very rare in the rectum. Of course this does not include presacral dermoids nor those occurring in the rectovaginal septum, which are not uncommon. Quenu[1] reports a case of dermoid of the rectum which perforated and was recognized by the protrusion of hair from the anus.

Benign Epithelial Tumors.— Adenomata or mucous polypi of the rectum do not include the polypi in the production of which the mucosa does not play a part, but simply forms a coating over them. At this time we are speaking of true *adenomatous polypi,* or *polypoid adenomata,* originating in the glandular elements of the mucosa. Not all adenomata form polypi; on the contrary, a considerable number of them appear as flat growths covering more or less of the rectal mucosa (Schwab[6]).

Mucous polypi usually occupy the lower third of the rectum and its posterior aspect, and are rarely larger than a pea or a hazel nut. They bear some resemblance to a raspberry.

As a rule, the growths do not give rise to *symptoms* other than those of a circumscribed catarrhal proctitis. In other instances, they prolapse through the anus and are then of course readily recognized.

The *treatment* consists of removal of the growth by excision or by the cautery snare; the latter method is preferable. Occasionally, tumors of this sort undergo malignant changes.

Polyposis recti is a term applied to multiple polypi which also cover more or less of the rectal and colic mucosa. The lesion is taken up in connection with those of the colon (p. 1228).

Papillomata of the rectum consist in groups of *villous polypi* attached to the mucosa of the rectum. The pedicles of the masses are likely to be very vascular. The epithelial constituents of the polypi present an appearance not unlike that of carcinoma. Quenu[1] regards the process as an initial step in cylinder cell carcinoma.

Clinically, the growths are not readily differentiated from mucous polypi. They bleed very readily; this circumstance should lead to

proctoscope examination and subsequent determination of the character of the process by means of microscopical examination of an excised specimen.

Carcinoma Recti.— Carcinomata of the rectum, in conformation with their origin, are cylinder cell cancers and, like those of the stomach, present special histological characteristics. These are discussed in connection with carcinoma in general (Part VI, chap. xvii).

For practial purposes, cylinder cell carcinomata of the rectum are divided into three groups:

1. *Carcinomata originating in the wall of the ampulla of the rectum* constitute about 70 per cent of the cases (Funke[7]) and are usually of the adenomatous type, either of the medullary or of the fibrous variety. In most instances, the lesion is primarily situated in

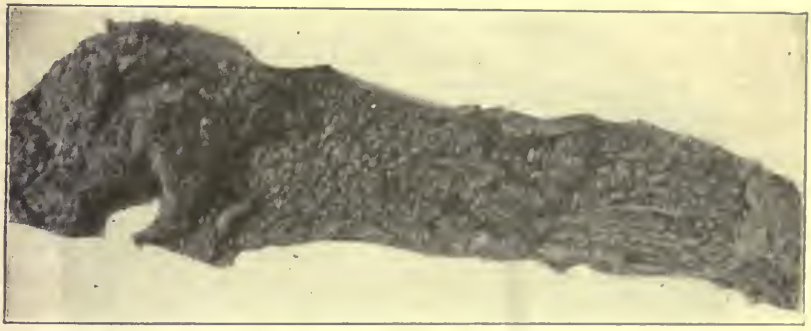

Fig. 956.— Polyposis of Colon Changing into Carcinoma.

the anterior wall of the ampulla just above the anal canal. After a time the process encircles the rectum.

2. The second group is that of the high *fibrinous carcinomata* (23 per cent, Funke[7]). These are located above the fold of Douglas in the pelvic colon and flexure. As a rule, the lesion consists of a relatively small (size of a walnut), hard, scirrhous tumor, which soon causes stenosis.

3. To the third group belong those which very early in the disease *infiltrate the greater part of the rectum.* These are usually alveolar or *colloid* carcinomata and comprise from 12 to 15 per cent of the cases. The lesion begins in the lower segment of the rectum and extends rapidly in all directions. In this way the entire organ is converted into a rigid tube filled with ulcerating masses.

Occasionally, carcinoma begins outside the rectum, where it arises

from displaced embryonic rests. In this event, the lesion is always located behind the rectum in the hollow of the sacrum and is easily mistaken for sarcoma.

Most carcinomata of the rectum (except the colloid) extend very slowly and do not penetrate the wall of the organ until late in the disease. When the latter occurs, the invader soon reaches the bladder, prostate, vagina, etc., in front, and the sacrum behind.

Aside from extension by continuity, carcinoma of the rectum spreads by the lymphatics and lymph nodes; but these are not involved early in the disease.

The *lymphatics of the rectum* are of surgical importance and are arranged in four groups, (1) those of the anal skin; (2) of the zona

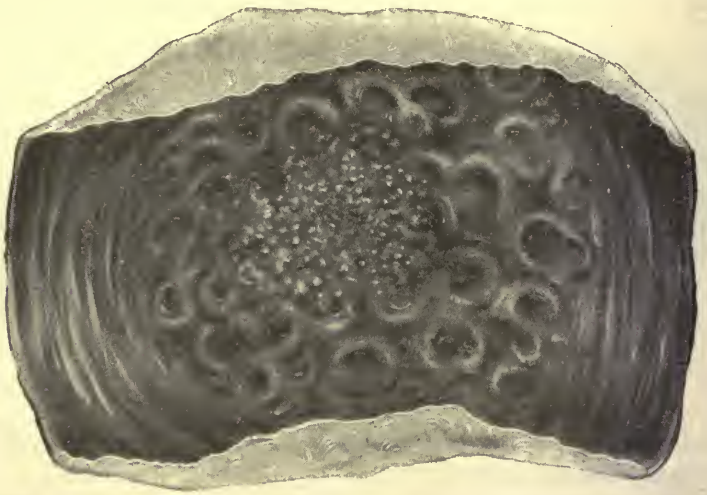

FIG. 957.— MEDULLARY CARCINOMA OF THE RECTUM.

intermedia of the portio analis; (3) of the zona columnaris of the portio analis; and (4) those of the pelvic rectum. The lymph vessels of (4) and (3) correspond to the course of the distribution of the superior hemorrhoidal vessels, which reach to the lower and of the columnae Morgagni. The lymph vessels of the intermediate zone communicate with the first and third systems. The pelvic anus does not contain any lymph nodes, while the pelvic rectum is richly endowed with them. In the extraperitoneal portion of the pelvic rectum the nodes lie to either side and follow the blood vessels in this situation. Several glands are also situated at the posterior aspect of the rectum and communicate

freely with those located between the layers of the mesentery of the colon.

The lymph vessels of the anal region drain into the glands lying in Scarpa's triangle; most of those of the zona columnaris and the pelvic rectum empty into the glands situated at the lateral walls of the extraperitoneal portion of the rectum, while a few communicate with those following the course of the median hemorrhoidal vessels (Quenu[1]). Finally, the lymphatics of the anal skin anastomose with those of the intermediate zone and of the anorectal region.

Nothing that has been said with regard to carcinoma in general (Part VI, chap. xvii) may be added to here with regard to *etiology*. The disease is not frequent. It is, perhaps, most likely to occur in situations subjected to chronic irritations.

The *symptoms* of carcinoma of the rectum are unfortunately not distinctive in the early stages of the disease.

The first symptoms to attract attention are those consequent to stenosis and ulceration. *Ulceration* of the growth, which occurs comparatively early, is at first attended with the expulsion of mucus and small particles of tissue and, later, of blood with the stool. The iritation caused by these elements is responsible for frequent emptying of the bowel. Bleeding occurs in 90 per cent of the cases and, as is to be expected, is least manifest in scirrhous carcinoma. On the other hand, in the medullary form of the disease, bleeding occurs early and is likely to be severe.

Symptoms of *stenosis* occur most frequently in connection with the scirrhous form of carcinoma, especially when the lesion is located high up; this is usually circular. Stenosis is frequently the first symptom. Early in the disease there is rather obstinate constipation, often attended with colicky pain and readily relieved by catharsis. The patient complains of dull pain and a feeling of fullness in the rectum and sacral region. Occasionally the symptoms of ileus develop. In many cases the latter are produced by invagination of an annular carcinoma into the lower rectum. Long periods of diarrhea alternate with the more persistent periods of constipation. The colloid form of carcinoma may also give rise to constipation, the infiltrated rectal wall being incapable of expelling its contents. Occasionally ulceration destroys sufficient of the obstructing gut to reëstablish its lumen. At this stage of the disease, the fecal mass is mixed with the products of the ulcerative process. The frequent defecation paralyzes the sphincters, so that incontinence develops, which ultimately may become complete.

At first the general health of the patient is not impaired. The constitutional effect of the disease does not become manifest until the tumor begins to disintegrate, and is due to the absorption of the products of putrefaction and the effect of stenosis. Thus, a condition of chronic sepsis is established which, together with pain, tenesmus, bleeding, and persistent diarrhea, finally closes the scene. In a certain number of cases perforation at the site of a high carcinoma causes peritonitis. A few cases die of hemorrhage, and in a certain number of cases death is caused by metastases. The latter occurs most frequently in connection with colloid carcinoma.

The *diagnosis,* when the growth is reached with the finger, is easy. Occasionally a case comes under observation when the process is in its early stages. In these instances the finger notes the presence of an indurated area or of a hard polypoid nodule. In these cases the diagnosis rests upon the microscopical examination of an excised sector of the growth. However, as a rule, digital examination reveals the presence of an insular, round or irregular ulcer with hard edges, sharply defined against the contiguous smooth mucosa. The edge of the ulcer is elevated above the niveau of its surroundings and is readily distinguished from the flat granulating surface of gonorrheal or luetic ulceration. Colloid carcinoma imparts its soft, nodular consistence to the finger.

Carcinomata located beyond the reach of the finger are not readily differentiated from luetic lesions. The former are characterized by almost normal mucosa below the growth. The diagnosis may be established by excision of a sector of the tumor through the proctoscope.

At the first glance, an invaginated carcinoma may present a confusing picture. The examination must be made after the invagination is reduced. It is a singular fact that in carcinoma located high up in the rectum, the walls of the lower segment do not lie in apposition, but gape widely. This may be noted in obstruction of the sigmoid from other causes, even in volvulus, and is a point well worth remembering. When, after intelligent employment of the various methods of examination, which include scrutiny of the stool, proctoscopy, the Röntgenogram and the microscopical examination, the case is still in doubt, exploratory celiotomy is justifiable.

Sarcoma usually begins outside the mucosa and appears in the form of a round soft tumor bulging into the lumen of the rectum.

The indications for *radical extirpation of the growth* have been much extended in the last fifteen years. A patient afflicted with a

carcinoma located above the fold of Douglas is no longer unqualifiedly condemned to death. Relief in this connection is made possible by the sacral route or by a combination of this route with the abdominal method of approach. This means that the height at which the lesion is located is not a deterrent as regards operative measures of relief; the limitations in this connection relate to the progress the disease has made with respect to the organs lying in radial relationship with the rectum, i. e., the sacrum, prostate, bladder, vagina, uterus, etc. The extent of the radial involvement is, in many instances, only discoverable by exploratory celiotomy; this permits of the immediate establishment of an artificial anus in case the technical problem is unsurmountable. In the other event, the abdominal approach permits of mobilization of the upper portion of the lesion, thus making extirpation of the growth from below easier. In not a few instances, apparently inoperable tumors become operable after an artificial anus has existed for a time.

The question as to whether a carcinoma of the rectum is susceptible of operative measures of relief is also influenced by the existence of lymphatic or organic metastases. When these are present radical extirpation is not permissible unless an invaginated growth causes ileus.

The preparation of the patient should be especially thorough. *Preliminary colostomy* is employed only when the growth has caused stenosis. The step interferes much with execution of the combined operation and should be avoided if this method is to be employed. When diversion of the fecal current is necessary, a *typhlostomy* is the least objectionable of the various methods in use. The fact that operations of this sort are always prolonged suggests the advisability of scopolinmorphin narcosis, supplemented by ether inhalation.

In *extirpation of the rectum* the patient is best *postured* in the so-called Depage[8] position. The patient lies on his stomach with hips elevated, i. e., a Trendelenburg position with the patient lying face down. Thus, when the pouch of Douglas is opened much of the abdominal contents becomes visible.

Partial excision of the rectum is practiced through the anus with the patient in the exaggerated lithotomy position. For the purpose the sphincters are dilated or sectioned posteriorly as far as the tip of the coccyx. The method rarely suffices; its employment is justified when the diagnosis of a carcinoma situated low in the rectum is made early in the disease.

Extirpation of a carcinoma, together with its ramifications can be accomplished only by *circular excision of the rectum* in which a greater

or lesser amount of the cylinder and, the regional glands, are removed. All other procedures are fallacious.

When the tumor lies in the region of the sphincter muscles, the latter must be removed. As a portion of the rectum is also removed, the operation is called *amputatio recti.* When a large sector of the rectum, together with the sphincteric area is removed, the term *extirpatio recti* is used; when the sphincters are preserved, the operation is termed *resection of the rectum.* The term *excision of the rectum* is applied by some surgeons to either of the methods.

The deep location of the rectum has made surgical attack upon it a difficult problem. For many years surgeons approached the organ by means of an incision made between the pubes and the coccyx — the *perineal route.* This method makes excision of the lower portion of the rectum possible, but gives little room for radical measures. At this point the scene changed and the rectum was approached from *behind* — the *dorsal method* — which has almost entirely displaced the perineal. In women it is feasible to remove the rectum through the *vaginal route.* Recently, the purpose has been accomplished by freeing the rectum through a celiotomy wound and completing the extirpation from below — the *combined method.*

The *perineal method* is employed for removal of carcinomata located in the ampulla, i. e., when the sphincters must be sacrificed. The patient is placed in the ventral, elevated pelvis position, and the anus is encircled by an incision 3 to 4 cm. from it; additional room may be obtained by splitting the perineum in the median line in front and behind to the anus. Thus the levator ani muscle is exposed and the divided branches of the inferior hemorrhoidal artery are tied. In front, the connections of the external sphincter, with the transversus perinei and the bulb, are divided; this separates the rectum from the infradiaphragmatic space. The exposed levator ani is divided behind, exposing the parietal pelvic fascia, which is pushed backward to the tip of the coccyx; the same step is executed on either side of the rectum. The pelvic fascia is next opened, thus making possible access to the pelvic rectal space. The fingers now enter the wound in front, and after the levator muscle has been recognized in this situation, it is divided in front of the sphincter ani. Next, the rectoprostatic fascia is opened on both sides and access to the space between the prostate and rectum is gained. The rectum is now attached to the prostate and bulb only by a narrow process of the rectoprostatic fascia; this is divided with the

knife "under the eye," care being taken not to injure the urethra or to enter the rectum.

In women, separation of the vagina from the rectum is more simple than is isolation of the latter from the prostate. Finally, removal of the rectum is accomplished in the manner described in connection with the dorsal method.

The *dorsal method* gives a much better exposure. A study of the sagittal section of the pelvis shows that the rectum from the anus to the coccyx lies in contact with soft parts, and that its exposure from behind is a simple matter. The evolution of the dorsal method of approach is presented by Rotter;[9] it received its greatest impetus from Kocher (1874), who resected the coccyx, and was more completely worked out by Kraske[10] (1885), who resected also a portion of the sacrum. The fear of injury to the sacral nerves and its consequences, has been shown to be groundless by the work of Hochenegg.[11] As time went on, it was demonstrated that simple resection of the coccyx and of the fourth sacral vertebra gave ample room for the purpose. Finally, the operation was modified so that instead of sacrificing the bone, the coccyx and a part of the sacrum are turned aside in the form of an osteoplastic flap, and in this way the operative field is exposed to a sufficient extent to permit of free access to the rectum, and after the resection is completed the flap is replaced. In 1911 Völker[12] showed that, by placing the patient in the Depage position, the sacral approach made visible the entire operative field. The technic employed by Tuttle[5] is as follows:

"An oblique incision is made from the level of the third foramen on the *right side of the sacrum* down to the tip of the coccyx, and extended halfway between this point and the posterior margin of the anus. This incision should be made boldly with one stroke through the skin, muscles, and ligaments into the cellular tissue posterior to the rectum; the latter is rapidly separated by the fingers from the sacrum, and the space thus formed, together with the wound, is firmly packed with sterilized gauze; a transverse incision down to the bone is then made at the level of the fourth sacral foramen, the bone is rapidly chiseled off in this line, and the triangular flap is pulled down to the left side, where it is held by the weight of a heavy retractor attached to it. At this point it is usually necessary to catch and tie the right lateral and middle sacral arteries.

"The first step in the actual extirpation of the rectum consists in isolating the organ below the level of the resected sacrum, so that a

ligature can be thrown around it, or a long clamp applied to control any bleeding from its walls (Fig. 958). If the neoplasm extends above this level and it is necessary to open the peritoneal cavity to extirpate it, one should do this at once, as it will be found much easier to dissect the rectum out by following the course of the peritoneal folds. By opening the peritoneum and incising its lateral folds close to the rectum, the danger of wounding the ureters is greatly decreased and the gut is much more easily dragged down. When the posterior

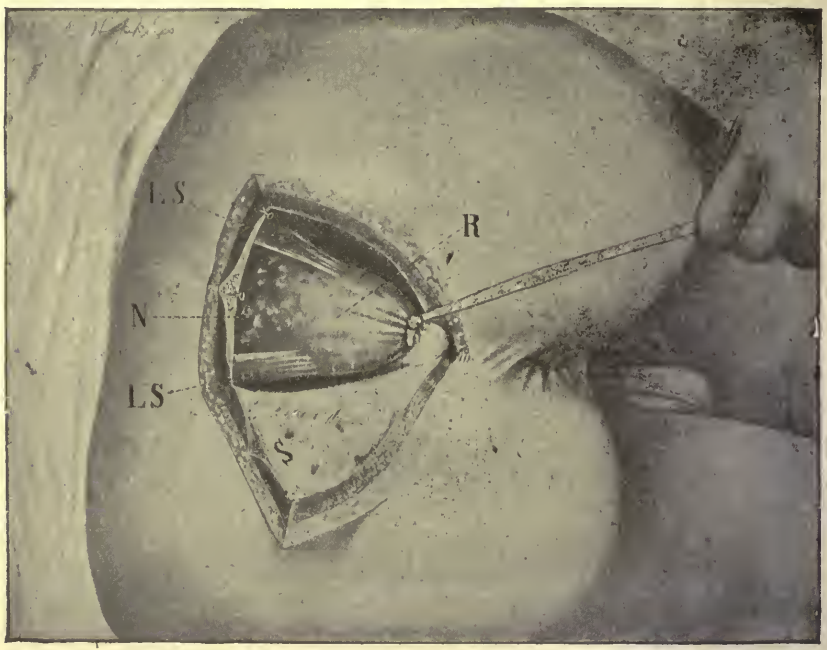

FIG. 958.— SECOND STEP IN BONE FLAP OPERATION.

R, Rectum; *N*, neoplasm; *LS*, lateral rectal ligaments; *S*, sacrum.

peritoneal folds or mesorectum are reached, the incision should be carried as far away from the rectum, or rather as close to the sacrum, as possible, in order to avoid wounding the superior hemorrhoidal artery and to remove all the sacral glands. The gut should be loosened and dragged down until its healthy portion easily reaches the anus or the healthy segment below the growth (Fig. 959). A rubber ligature or strong clamp should then be placed upon the intestine about 1 inch above the neoplasm, but should never be placed in the area involved

by it, for in so doing the friable walls may rupture and the contents of the intestine may be poured out into the wound. As soon as the gut has been sufficiently liberated and dragged down, the peritoneal cavity should be cleansed by wiping with dry sterilized gauze, and then closed by sutures, which attach the membrane to the gut. By this procedure the entire intraperitoneal part of the operation is completed and this cavity is closed before the intestine is incised. After this is done, the gut is cut across between two clamps or liga-

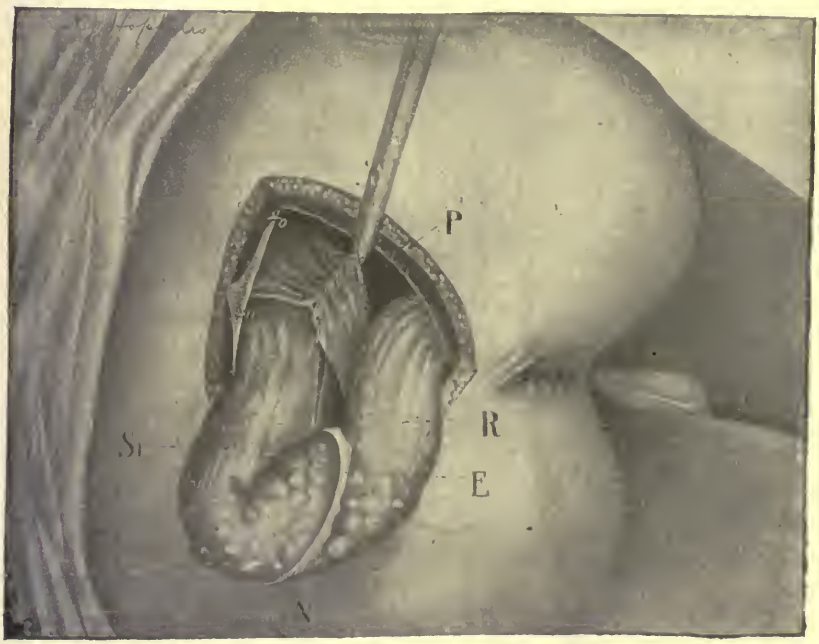

FIG. 959.—FOURTH STEP IN BONE FLAP OPERATION.

R, Rectum.

tures above the tumor. The lower segment containing the neoplasm may then be dissected from above downward in an almost bloodless manner until the lowest portion is reached. It is much more easily removed in this direction than from below upward, and there is less danger of wounding the other pelvic organs. If the neoplasm extends within 1 inch of the anus, it will be necessary to remove the entire lower portion of the rectum. If, however, more than 1 inch of perfectly healthy tissue remains below, this should always be preserved.

Having removed all of the neoplasm, if 1 inch or more of healthy gut remains above the anus, one should unite the proximal and distal ends of the gut by the end to end suture. Posterior fistula follows in almost every case, but it generally heals spontaneously, and need give no serious concern.

"Where there is less than 1 inch of the rectum left below, and the gut can be easily brought down to the anus, it is well to dissect off the mucous membrane from the latter organ, invaginate the upper end of the intestine through this, and suture it to the skin outside. The gut is not sutured to the margin of the anus, but entirely outside of it, in order that the fecal passages will not come in contact with the line of union between the freshened portion of the anus and the peritoneal surface of the gut which has been dragged down. No tension should be employed in bringing the gut down to position. After it has been fixed in place, a large, silk, anchoring suture is placed in the mesorectum about 3 inches above the anus and attached to the skin outside the lower angle of the wound, in order to prevent retraction of the gut and tension on the sutures. In some instances in which, contrary to expectations before the operation, we are unable to reunite the ends of the intestine or to bring the proximal end down to the anus, it is necessary to attach the latter at a higher level of the wound, thus forming what is known as a sacral anus. All oozing is checked by hot compresses, and the concavity of the sacrum is packed with a large mass of sterilized gauze, the end of which protrudes from the lower angle of the wound; this serves to check any oozing and also furnishes a support to the bone flap after it has been restored to position. Finally, the flap is fastened back in its original position by silkworm gut sutures, which pass deeply through the skin and periosteum on each side of the transverse incision. Suturing the bone itself is not necessary. The lateral portion of the wound is closed by similar sutures down to the level of the sacrococcygeal articulation; below this it is left open for drainage. A large drainage tube is carried up through the gut beyond the line of intestinal sutures, and the whole is dressed with sterilized absorbent gauze held in position by adhesive straps and a firm T-bandage."

The *vaginal method* of resection of the rectum was first used by Desguins.[13] The procedure may also include removal of the sphincters. The technic of the measure is as follows:

The vagina is dilated with broad retractors, the cervix is drawn down, and the pouch of Douglas is opened by a transverse incision

just below the cervical juncture. The small intestines are then pushed upward out of the way, and the peritoneal cavity is packed with large laparotomy pads. The rectovaginal septum is then divided by a vertical incision in the median line, extending from the first incision down to the margin of the anus, and including the external sphincter. The vaginal wall is then dissected from its attachments to the rectum, thus exposing this organ in its entire length, enabling one to examine it and drag down the sigmoid flexure almost at will. At this point the anterior rectal wall is divided up to the lower border of the tumor, and the gut incised transversely 1 inch below the lower limits of the growth, the incision being carried into the retrorectal tissue. The proximal end of the gut is then grasped with forceps, which close it, and by the use of curved scissors it is separated from its posterior attachments as far as the promontory of the sacrum, or at least sufficiently far for the bowel to be drawn down until its healthy portion reaches the lower segment without undue tension. The gut is then amputated above the growth, and the upper and lower segments are united end to end by silkworm gut sutures. These sutures are passed from within outward, the knots being tied upon the inside, and the ends left long to facilitate their removal. The wound in the anterior wall of the rectum is closed in the same manner, and the ends of the sphincter brought together by buried catgut sutures. After the laparotomy pads have been removed, the peritoneal wound is closed with a continuous catgut suture. A large drainage tube is introduced through the anus above the point of anastomosis and sutured in position, the vagina and external parts being dressed with sterile gauze.

The *combined and the abdominal methods* are employed in cases in which the tumor is located too high for the dorsal method and too low for the abdominal.

The idea of the combined method originated with Volkmann (1877) and was worked out by Chalot,[14] Quenu,[1] Chalier,[15] Rotter,[9] and others. The *abdominal portion* of the operation is executed in the manner indicated in connection with resection of the sigmoid (p. 1951). (See also Hochenegg,[11] Rehn,[16] Mayo,[17] Ali Krogius[18]). The *dorsal portion* of the operation is performed in the manner stated in connection with the dorsal route immediately above. It is, of course, occasionally feasible to combine the abdominal with the transanal or perineal route. However, the measure presents considerable technical difficulty as regards the lower part of the operation.

A number of surgeons, notably Quenu,[1] always remove the sphincters and establish an *inguinal* colostomy. In these instances the upper end of the rectum is deligated and ultimately delivered through the perineal opening.

The purely *abdominal operation* is carried out along the lines of a sectional colectomy, and is employed only in cases in which the lesion is located well above the pouch of Douglas (Göpel[10]).

In the *after treatment* it is important that the patient be postured so as to provide for drainage. Iodoform gauze tamponade is permitted to remain *in situ* from eight to ten days. The bowels must not be permitted to move during this time. When this finally takes place, the wound surfaces are thoroughly cleansed. Catheterization is usually necessary for several days following the operation.

Excision of the rectum is a difficult operation. A fatal outcome is due primarily to shock, and secondarily to infection. The latter often occurs late from contact with feces. In some cases iodoform intoxication is undoubtedly responsible for an unfavorable outcome (p. 72). As in all extensive operations, the result is often marred by the development of pneumonitis. A statistical review, collected by Rare, with regard to the mortality is presented by Rotter.[9]

The surgeon must bear in mind that the susceptibility of carcinoma of the rectum to operative measures of relief no longer depends upon its location, and that in all cases, the dorsal, the combined, or the abdominal methods of approach make the lesion accessible to surgical measures. It is also true that, of these, the method which permits of visualizing the operative field is followed by the least number of deaths and by the best functional results.

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PART XVIII

THE SURGERY OF THE FEMALE ORGANS OF GENERATION

CHAPTER I

CONGENITAL AND ACQUIRED MALFORMATIONS

Hermaphroditism.— Hermaphroditism is a condition in which the male and female organs of generation apparently or actually exist in the same person. Although this condition of affairs is common in many forms of lower plant and animal life, the actual existence of true hermaphroditism in the human, where complete generative organs of both sexes are present, may be regarded as doubtful, despite the fact that it has been reported.

Pseudohermaphroditism is divided into the *gynandrous* and *androgynous*. In the former the external genitals simulate the male and the general development and facial expression is masculine, while in the latter the general characteristics and genitals are feminine.

The *treatment* consists in actually determining the sex and bringing the child up accordingly. If doubt exists, exploratory celiotomy to determine the character of the internal genitalia is permissible. Operative correction of the external genitalia may then be made in accord with these findings.

Malformations.— According to Bland,[1] *congenital malformations* are due to (1) complete failure of development; (2) rudimentary or incomplete development; (4) vicious development; (5) precocious development of the ducts of Müller.

Acquired malformations are most frequently the result of inflammatory processes with the formation of adhesions and occasionally occur in connection with imperforate hymen, pregnancy, operative measures, and chemical or thermal burns. Of these, the most important are discussed here.

In *imperforate hymen* the consequent accumulation of menstrual blood is called *hematocolpos;* when the uterus is filled the term *hematocolpometra,* and when the salpinx is also filled the term *hematocolpometrasalpinx,* are used. The condition may also occur in connection with *stenosis* or *atresia* of the *vagina.* In accord with the character of the accumulation, the terms *hydrocolpos, pyocolpos* or *gas colpos* are used.

The *treatment* consists in the free incision of the obstructing membrane, drainage of the genitals, and light tamponade of the vagina.

Atresia and stenosis of the vagina may be *congenital* and are found at different parts of the canal. However, they most frequently occur near the outlet, the result of imperforate hymen. The *acquired* form follows injury, labor, burns, etc., or is caused by neoplasms, and the cicatrices sequential to ulceration (pessary), drainage of the *cul de sac* or other operative procedures.

Complete absence of the vagina is usually associated with absence of the uterus, the fallopian tubes, and, occasionally, the ovaries. However, the vagina alone may be absent.

The *operative treatment* consists in making an incision between the urethra and rectum, followed by blunt dissection of the structures between them until the peritoneum is reached. The cavity thus formed is lined by flaps dissected from the labia majora and minora — if large enough — and from the inner sides of the thighs. Vaginal plugs are introduced to avoid cicatricial contraction. Carl Beck[2] makes a transverse incision above the symphysis and dissects downwards between the bladder and the peritoneum, making a counter incision between the urethra and rectum. Flaps are obtained from the inner side of the thighs and are pulled upwards by a forceps which is introduced into the canal through the abdominal incision. The suprapubic wound is closed and the perineal cavity is packed tightly with iodoform gauze so as to press the flaps against the sides of the canal. Baldwin[3] recommends using a loop of intestine for the lining of the new vaginal canal, while Makenrodt[4] advises transplantation of vaginal mucous membrane, removed from patients operated upon for prolapsus uteri.

Double vagina (Fig. 961) is a not uncommon deformity. The canals may be more or less equal, or one may be small and the other fully developed.

The *treatment* consists in section of the septum by cutting it and quilting the edges by a continuous suture or employing the actual cautery. Iodoform tampons or plugs are inserted.

Malformations of the Uterus.— Absence of the uterus is exceedingly rare and is associated with absence of other organs of reproduction. The uterus may exist as a *rudimentary body* or may be *infantile in structure.* The latter (if it does not subsequently develop) becomes a functionless organ. The womb in this condition

is decreased in size in all its dimensions, with a greater development of the cervix than of the body.

In *double uterus* (Fig. 962) the organ is divided by a septum which may be limited in extent or may divide the organs into two distinct cavities; or the uterus may appear as two lobes attached to a single

FIG. 960.— IMPERFORATE HYMEN (Kelly).

cervix. The uterus may appear as two cornua attached to one cervix (*uterus bicornis*), one horn developed and the other atrophied (*uterus unicornis*). Accessory uteri have been described.

MALFORMATIONS OF THE TUBES AND OVARIES.— Absence of the fallopian tubes is a rare condition and is associated with absence of other organs of the genital apparatus. The absence of one tube is usually

coincident with unicornate uterus. The tubes may exist as rudimentary structures. Accessory openings, absence of the canal, double canal, and accessory tubes are rare conditions.

Absence of the ovaries is the least frequent malformation of the female genitals and may exist without other derangement of the genitals. Absence of the ovary usually occurs with absence of a kidney on the corresponding side. Rudimentary ovaries occur occasionally, while accessory ovaries are frequently present.

Vaginismus.— Vaginismus may be *defined* as a painful tetanic spasm of the muscles surrounding the vaginal outlet. It appears in two forms, the *inferior,* attended with spasm of the vulvovaginal muscles; and the *superior,* attended with spasm of the levator ani muscles.

Fig. 961.— Double Vagina (Kelly).

It occurs most frequently in young, nervous, hysterical brides as a distinct neurosis, though the condition is occasionally found in multiparae. In some instances it is secondary to an unyielding hymen, fissures, and abrasions of the vaginal outlet, urethral caruncle, and anal fissure. Forced, ill directed attempts at coitus, a large penis, and premature ejaculation may induce extreme nervous irritability in the female with resulting vaginismus.

Symptoms: In inferior vaginismus, attempt at intercourse is attended with painful muscular contractions which render the sexual act impossible. In the superior type entrance into the vaginal outlet is accomplished, but upon partial penetration the penis meets the

resistance of the levator muscles. If this spasm is overcome, the penis above the corona is encircled by the levator fibers, resulting in the temporary painful vaginal imprisonment of the penis (penis captivus).

In the *treatment* the patient is examined in narcosis with the view of determining the cause as stated above. When none is discovered, the sphincter vaginae is forcibly stretched and a snug fitting glass plug is introduced into the canal. Existing lesions are treated on general principles.

In intractable cases (without a discoverable causative lesion) it is permissible to excise the hymen and forcibly dilate the vaginal outlet. This is followed by a V-shaped incision extending deeply into both vaginal sulci halfway through the perineum. Later, the vagina is systematically dilated with bougies.

Surgery of the External Genitals.— Traumatism to the vulva, because of its protected position is not common. It occurs in connection with a fall, kick, or blow, or as the result of the use of instruments. Injuries during childbirth are of course the most common cause.

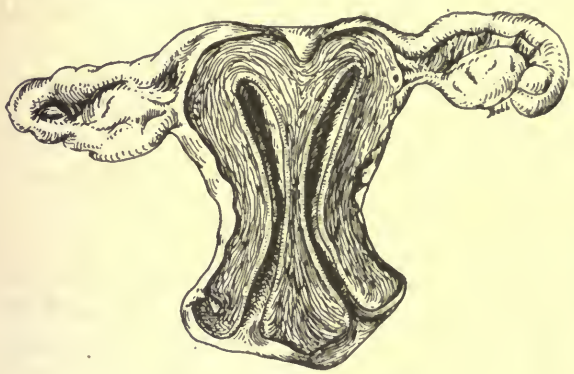


FIG. 962.— DOUBLE UTERUS.

Incised wounds are characterized by severe bleeding, especially when they occur in cases of preëxisting varicosities (during pregnancy).

Contusion of the vulva is likely to be attended with massive subcutaneous bleeding and often forms a pudendal hematoma of considerable size, involving both labia.

The *treatment* in restricted infiltrations is purely symptomatic. Large hematomata are best opened and the contents allowed to escape; the residual cavity is packed with iodoform gauze; infection, especially in the after care, must be carefully guarded against.

VARIX.— Varix (varicose veins of the vulva) is often associated with a similar condition of affairs involving the veins of the lower extremities. The lesion is caused by the presence of intrapelvic tumors

or chronic inflammatory processes, by constipation and by prolonged maintenance of the erect position. The condition is markedly aggravated by pregnancy. The veins are easily traumatized.

In the *treatment,* the underlying cause should be removed. If this fails, the varicosities may be excised.

Vulvitis.— Vulvitis is an inflammatory condition of the lining of the vestibule, due to uncleanliness, to the irritation of vaginal secretions, and to invasion by the gonococcus or other bacteria.

The process is attended with *severe itching,* pain, and burning, especially on walking. The labia are swollen, tender and hyperemic and are covered by mucus or mucopurulent secretion. The sebaceous glands and hair follicles are often invaded in the form of a pustular or papillary inflammation (*follicular vulvitis*).

The *treatment* consists in rest in bed, which is very important, the frequent use of soothing douches (zinc) and sitz baths. (Gonorrheal vulvitis is discussed elsewhere, p. 2239.)

Bartholinitis.— Bartholinitis is a term applied to inflammation of the vulvovaginal glands. It is usually gonorrheal in character and is associated with a similar process in the vagina.

The lesion appears in the form of one or more abscesses located deeply in the labia.

The *clinical picture* is that of vulvitis plus abscess.

The early *treatment* is that of vulvitis. When suppuration is manifest the focus is freely incised and drained — preferably with iodoform gauze. In a certain number of cases the gland remains chronically inflamed and must be extirpated.

Cysts of the glands of Bartholin are really retention cysts. They appear in the form of globular tumors the size of a cherry and may increase to that of a walnut or of a lemon.

The *symptoms* are those of a low grade of inflammation with tumor formation. In a certain number of instances a cyst of this sort ruptures and spontaneous healing follows. As a rule, however, it is necessary to *extirpate the cyst* together with its entire wall.

Abscess of Skene's Glands.— Abscesses of Skene's glands occur in connection with gonorrheal urethritis. After the acute symptoms have subsided, the gonococcus remains in the ducts and gives rise to the formation of a small abscess located on either side of the urethra about ⅛ of an inch from the meatus.

The abscess appears in the form of an oval, fluctuating, painful

tumor which encroaches upon the wall of the vaginal vestibule. Pressure on the mass causes pus to ooze from the urethra.

The *treatment* consists in incision and drainage of the abscess.

Hypertrophy of the labia minora, which interferes with function, demands ablation of the abnormal tissue. A similar condition of the clitoris is treated by amputation (clitoridectomy).

Clitoridectomy is occasionally necessary for the relief of tuberculosis or malignant disease in this situation.

Fig. 963.— Abscess of Skene's Gland.

Hypertrophy of the prepuce or its adherence to the glans clitoris is occasionally responsible for reflex nervous symptoms.

From a surgical standpoint these conditions are important. The hypertrophied prepuce is ablated and adhesions are divided. These measures are promptly followed by relief.

Elephantiasis of the vulva, other than that of parasitic origin (p. 960), is probably due to a chronic inflammatory process, such as lues, which obstructs the lymphatic circulation. The lesion is that of a simple hyperplasia and hypertrophy of apparently normal skin and the connective tissue.

The hypertrophy is attended with dull pain, itching, and later with ulceration. In severe cases, coitus, locomotion, and defecation are more or less interfered with.

Removal of the redundant tissues is indicated. Antiluetic treatment is permissible.

Condylomata of the vulva present themselves in two forms — the *non-specific* and the *specific.*

Non-specific condylomata are warty growths (hard papillomata) which are single, multiple, and often pedicled.

Specific condylomata occur in the form of venereal warts (*gonorrheal condylomata*) developing on an inflammatory base. They may occur singly, may be multiple, or may be present as confluent cauliflower-like masses. The excrescences are branched or pointed in form, of a grayish white or red color, and are usually bathed in secretion.

Luetic condylomata (condylomata lata) are similar to the mucous patches seen elsewhere. They appear as soft grayish warty excrescences with broad bases.

The *treatment* of hard papillomata consists in excision. Luetic condylomata are cleansed and kept covered with ung. hydrarg. This is supplemented with the constitutional treatment of the cause. In the other forms of condylomata, cleanliness and drying of the parts with powders usually accomplish the purpose. In extensive cases the condylomata may be excised and the stump cauterized.

FIG. 964.— CONDYLOMATA OF THE VULVA.

Lupus of the vulva, a very rare condition, presents itself in the form already described in connection with lupus of the skin (p. 430).

Urethral caruncle is an inflammatory hypertrophy of the urethral mucosa, which is very vascular, cherry red in color, and usually pedunculated. The lesion may be single or multiple, and may vary in size from that of a rice kernel to that of a hazel nut. The process may be divided into the simple and the neuromatous.

The *symptoms,* in the *simple variety,* are vague, and the lesion may be accidentally discovered. In the *neuromatous form* the mass is richly supplied with nerve fibers, which are responsible for great pain on walking or urination and for more or less serious reflex nervous disturbances. The latter are perhaps best characterized as hysterical. Bleeding occurs in both types of the lesion.

The simple type demands *treatment* only when bleeding occurs, in which event the growth is amputated and the stump cauterized. The

treatment of the neuromatous form is similar to that indicated for the relief of the simple form.

Hydrocele is taken up in connection with hernia (Part XVI, chap. xii). See also Regnoli.[5]

Benign tumors of the vulva are uncommon. Of these, atheromata, lipomata, and fibromata are most frequently found. See Tumors, Part VI, chap. xvii.

Malignant tumors of the vulva do not possess regional peculiarities.

Sarcomata are most likely to be of the melanotic type and grow very rapidly. *Early extirpation* holds out the only possibility of relief.

Carcinoma of the vulva develops most often at the inner side of the right labium minus close to the clitoris. It appears in the form of a nodular infiltrate, with a tendency toward the formation of epithelial pearls and cornification. Ulceration occurs early, although the infiltrate, as such, also extends very rapidly. In other regards the lesion is similar to that of superficial carcinoma in general (Part VI, chap. xvii). Lymphatic metastases appear promptly.

Operative removal of the process, as is the case in all carcinomata, must be radical. The inguinal glands must be removed in all instances.

Surgery of the Vagina.— Foreign bodies in the vagina include a vast variety of articles, accidentally introduced, left there in the act of masturbating, or used for the purpose of inducing labor. Occasionally, portions of surgical instruments (needles, sounds, etc.) or of therapeutic apparatus are inadvertently left in the vaginal canal.

The *disturbances* depend upon the character of the foreign body. Sponges and tampons give rise to leukorrhea, and, later, to a fetid discharge, and may be mistaken for sloughing polypi, carcinomata, etc. A pessary may remain *in situ* for a long time without provoking disturbances, and then finally cause erosion and ulceration of the vaginal wall. In some instances, pressure ulceration extends into neighboring organs (bladder and rectum).

The *treatment* consists in removal of the foreign body and the care of the ulcerative process.

Non-puerperal injuries of the vagina are not common. They are most often produced in connection with rape or coitus, though they may also occur in the same way as injuries to other portions of the body are produced.

Surgical repair in this class of cases is carried out on general principles. Healing is often interfered with because of infection, the result of contamination of the wound surfaces with urine and feces.

This invites frequent cleansing and the use of the catheter. The bowels are best emptied by enema, which permits of immediate cleansing of the injured parts. Rapid aseptic repair obviates sequential loss of function, the outcome of cicatricial contraction. The latter is very likely to follow prolonged suppuration.

Puerperal Injuries.— Puerperal injuries of the pelvic outlet (according to Hirst[6]) are caused by the following: (1) a relative disproportion in the size of pelvic outlet and the fetus; (2) rapid expulsion of the fetus, anticipating the gradual dilatation of the maternal parts; (3) an abnormality in the mechanism of labor, where the presenting part is pushed back against the center of the perineum, preventing its propulsion forward under the symphysis pubis.

The injuries thus produced vary from a slight or superficial, to a deep or complete tear involving the sphincter ani muscle. Laceration of the levator ani muscles on one, or both, sides may occur with or without involvement of the vaginal wall. A complete perineal tear is usually associated with involvement of the levator muscles.

Later, a hernial protrusion of one or more pelvic organs through the relaxed vaginal orifice occurs in accord with the extent of the laceration and length of time after the injury. When the posterior vaginal wall bulges, the condition is known as a *posterior colpocele;* when the rectal wall is also protruded, the term *rectocele* is employed. A prolapse or protrusion of the anterior vaginal wall or roof is termed a *cystocele.* If the prolapsus continues, the uterus is dragged down; this is termed *prolapsus uteri,* and complete protrusion of the uterus, together with the vaginal walls, is termed *procidentia.*

The *symptoms* depend upon the extent of the laceration and its duration. At the time the tear occurs, bleeding is often severe, pain is inconstant. As time goes on and cicatrization is complete, the patient complains of dragging pain, backache, and more or less disturbance in urination.

The *treatment* of a primary perineal tear is of great prophylactic value. The repair of postpuerperal lacerations is divided into primary, intermediate, and secondary.

In the *primary operation* the cervix is sutured first, interrupted chromic gut sutures being used for the purpose. Frayed tissue is removed from the vaginal tears; the anterior one is sutured first and the torn sulci of the perineal rupture are repaired next by deep sutures made to include the separated and torn fibers of the levator ani muscles; these sutures are not tied until all are properly placed.

The superficial perineum is approximated with sutures placed sufficiently deep to include the retracted edges of the transversus perinei muscles.

In *complete tears* involving the rectum, the latter is repaired first, either by interrupted sutures passed through all coats of the rectum

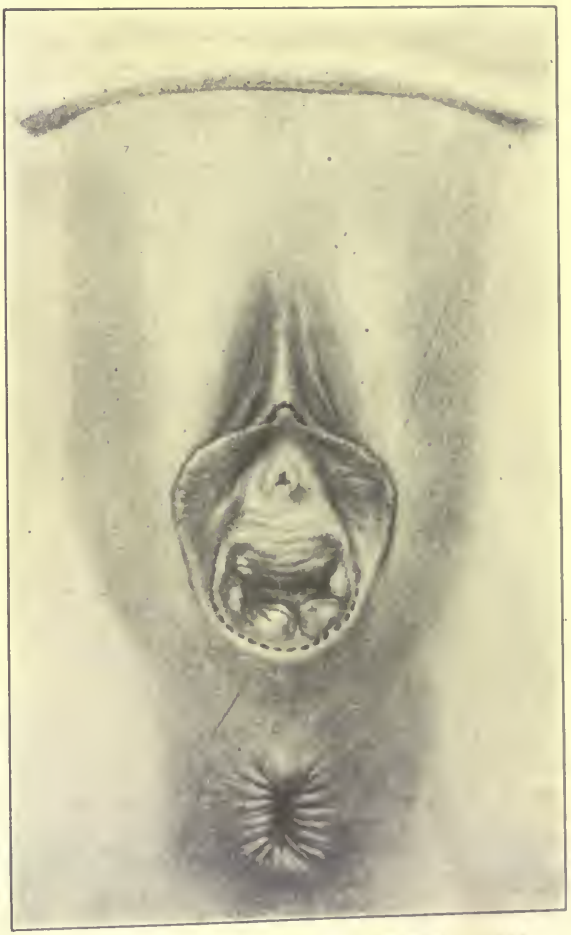


FIG. 965.— OUTLINE OF INCISION ON THE VULVAR MUCOSA (Sturmdorf).

and tied within its lumen, or by interrupted (or continuous) sutures which include the margin of the torn rectal wall, excluding its mucous coat. These sutures are tied from the vaginal side. The anal margin

and the sphincter ani are next united with interrupted sutures. A second, reinforcing layer of sutures may be placed in the rectal wall. The vaginal laceration is repaired as described above.

The *intermediate repair* is made from twenty-four hours to two weeks after the initial injury. The measure is employed when the primary operation is contra-indicated for one of the following causes: postpartum hemorrhage, virulent infection, eclampsia, severe shock following difficult delivery, and devitalization of tissue from pressure.

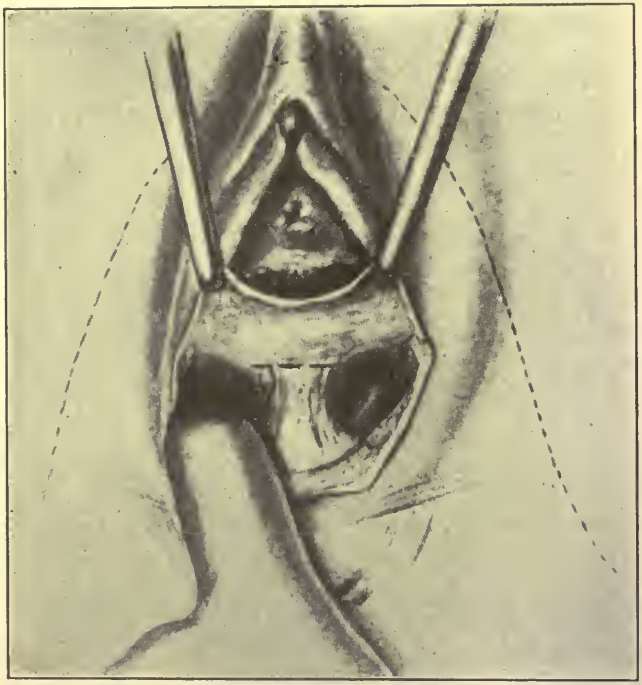


FIG. 966.—ISOLATION OF THE LEVATOR MUSCLES (Sturmdorf).

Denudation of the lacerated tissue is rarely required; rough sponging is all usually that is necessary, although scraping with a curet or knife may be employed. Ragged wound edges are trimmed, and the repair is made as in the primary operation.

The *secondary operation* consists in plastic repair of the torn parts. Numerous operative measures are employed for the purpose. These include the technic used by Hegar,[7] Emmet,[8] Ristine,[9] Tait,[10] Haynes,[11] and Babcock.[12] The methods may be divided into two main classes,

the *denudation* (Hegar[7] and Emmet[8]), and the flap splitting and resection operations (Ristine[9] and others). The operation (levator myorrhaphy) most widely employed at this time is described by Sturmdorf[13] as consisting of the following steps:

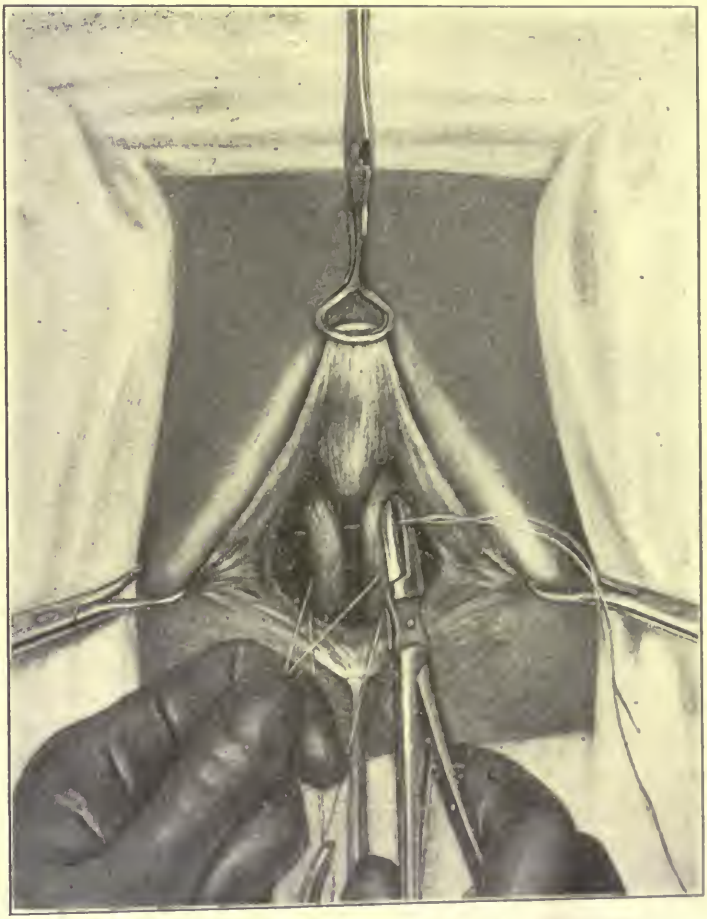


FIG. 967.—ENCIRCLING THE LEVATOR MUSCLES (Sturmdorf).

"I. Elevation of a thin, almost translucent triangular flap of mucosa from the posterior vaginal wall.

"II. Exposure and mobilization of the puborectal levator shanks.

"III. Sutural readjustment of the muscle.

"IV. Coaptation of the vaginal flap.

"V. Closure of the superficial perineal flaps.

"An incision is made on the vulvar mucosa (Fig. 965) from corresponding points just outside the lateral hymenal edges to the mucocutaneous center of the posterior commissure. This triangular surface

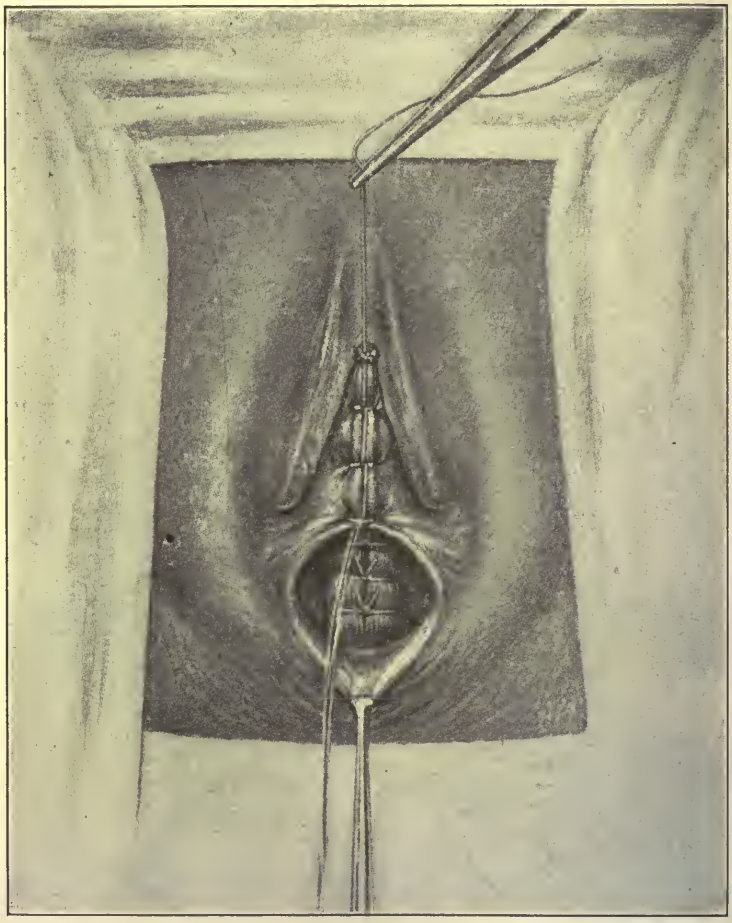


FIG. 968.— APPROXIMATION OF THE EDGES OF THE MUCOUS MEMBRANE FLAP (Sturmdorf).

is carefully elevated from the underlying tissues by dissection up to its point, producing a flap of vaginal mucosa only. The exposed submucous surface leads directly into the normal line of cleavage between the two levator sheaths. Both muscles are exposed by blunt dissection

with the fingers without injury to the sheath covering them (Fig. 966). Now, three to four interrupted, forty day, chromic gut sutures coapt the muscle, being passed from side to side entirely around both muscles, but not through them (Fig. 967). Care must be taken to avoid including the rectocele (if present) in a suture. In closing the vaginal flap no mucosa is removed. The transverse wound is converted into a perpendicular slit by a continuous suture, which ends at the lowermost

FIG. 969.— INVERSION OF FLAP OF VAGINAL MUCOSA.

Insert: *A*, Inverted flap; *B*, levator ani; *C*, superficial perineal coverings (Sturmdorf).

hymenal caruncles. The cone thus formed (Fig. 968) is inverted upon itself, tucked into the vagina and snugly applied to the posterior surface of the reunited levator ani muscle (Fig. 969). The levator fascia and superficial layers of the perinei muscles and the skin are united by a single or double layer of interrupted sutures."

CYSTOCELE.— Operative measures for the relief of *cystocele* are as numerous as those for repair of the perineum (Bumm,[14] Liepmann,[15]

Martin,[16] Hegar,[17] Frank[18]). Of these, the *anterior colporrhaphy* practiced by Frank[18] is perhaps most widely employed. It may be described as follows:

The cervix is delivered with a volsella forceps and the vaginal mucosa grasped with a small forceps ½ cm. below the urethra. A vertical incision is made between these instruments through the mucosa only; the latter is separated from the bladder (from below upward) for ¼ cm. along both sides of the incision. At the bottom of the incision the vesical ligament which connects the bladder to the cervix is cut. The gauze covered finger frees the lower margin of the bladder from the cervix by stripping upward and backward, exposing the pubovesical ligaments (Fig. 970). The vaginal mucosa is further separated laterally and the bladder pushed up well above the peritoneal reflexion, especially at the sides. Interrupted chromic gut sutures are passed from pillar to pillar, catching the cervix behind. Care must be taken to avoid including the urethra in the upper suture. The sutures are tied. The inverted V-shaped fascia at the upper end of the denudation close to the urethra is approximated by a transverse mattress suture. The remaining weak portion of unreinforced bladder is closed by interrupted chromic sutures. Excess and redundant vaginal membrane is cut away and the edges of the mucosa are united with interrupted silk sutures.

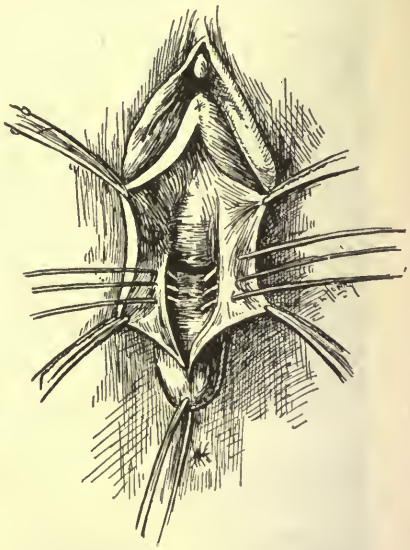


FIG. 970.— CYSTOCELE OPERATION (Frank).

The pubocervical ligament is exposed. The cervical sutures are introduced.

For the relief of cystocele with uterine prolapse the operative method employed by Wertheim, as modified by Watkins,[19] would seem to meet the indications.

The anterior vaginal wall is separated from the cervix by a semilunar incision and split, and the bladder is separated from the vagina and uterus. The uterovesical peritoneal fold is incised, the uterus is delivered through the opening, and the vesical peritoneal edge is

sutured to the corresponding surface on the posterior wall of the uterus. A suture is then passed through the vaginal flap below the urethra, through the body of the uterus posterior to the fundus, and out through the opposite vaginal flap (Fig. 971). At the same level a second suture is passed parallel to the first and both are tied, and the vaginal flaps drawn over the uterine body.

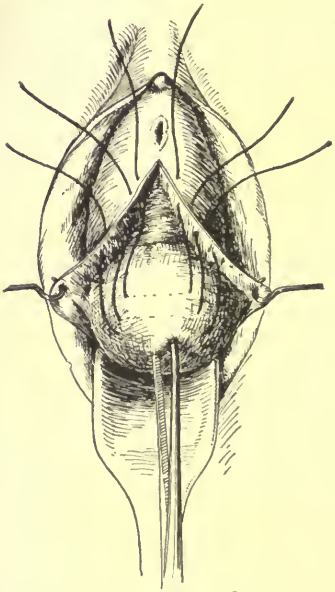


FIG. 971.— CYSTOCELE OPERATION (Wertheim-Watkins).

Suture of body of uterus to anterior vaginal wall.

FISTULAE OF THE FEMALE GENITAL TRACT.— Fistulae of the female genital tract are classified by Fisher[20] on an anatomical basis as follows:

Urethra.
- Urethrovaginal.

Bladder.
- Vesicovaginal.
- Vesico-uterine.
- Vesico-uterovaginal.

Ureter.
- Ureterovaginal.
- Ureteco-uterine.

Fistulae of the female genital tract are *caused* by pressure necrosis during labor, carcinomata, and chronic ulcerative processes.

The *diagnosis* of the character of the fistula is made by careful examination. In the urethrovaginal type the urine is retained until voluntarily discharged, but, with an intact pelvic floor or obstructing cicatricial bands, part of the urine may find its way to the upper portion of the vagina and may be discharged involuntarily, thus simulating a ureteral or vesical fistula. Careful examination with a speculum and probe usually makes the differentiation. A vesical fistula is recognized by sight or touch, and in some cases a sound may be introduced through the urethra and brought out through the vicarious opening. Placing a patient in the knee-chest position and retracting the posterior vaginal wall with a Sims' speculum may bring an otherwise imperceptible fistula into view. To differentiate vesical from ureteral fistula, milk or an analin dye solution is injected into the bladder. If no colored fluid appears, the fistula is ureteral; if vesical, the fluid exudes from the fistulous opening. The test is also

applicable in differentiation between vesico-uterine and uretero-uterine fistulae.

A fistula secondary to tuberculosis is inoperable. Fistulae heal either primarily or by granulation. Most small fistulae heal spontaneously; in the larger ones the vicarious opening is gradually reduced. Complete section of a ureter never heals spontaneously; an opening in its side may.

The *treatment* of fistulae is directed toward the maintenance of cleanliness by frequent douching. Thus, small fistulae often heal in

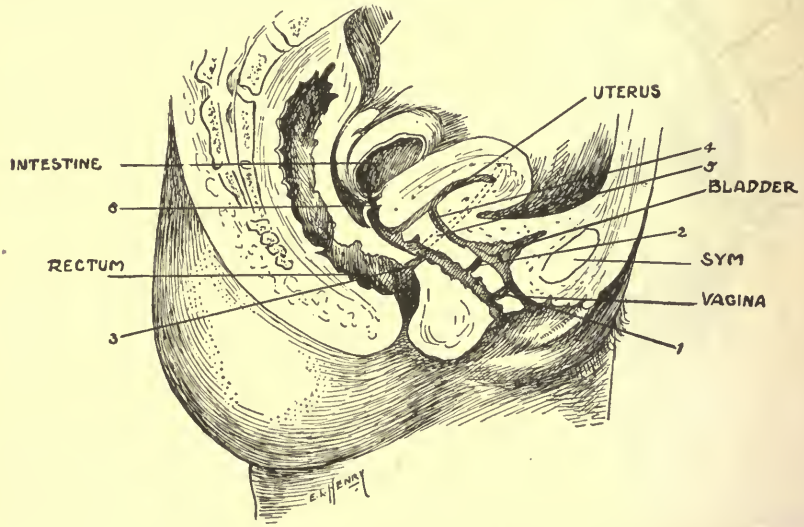


FIG. 972.—FISTULAE OF THE GENITAL TRACT.

1, Urethrovaginal fistula; 2, vesicovaginal fistula; 3, rectovaginal fistula; 4, vesicouterine fistula; 5, ureterovaginal fistula; 6, intestinovaginal fistula (after Gilliam).

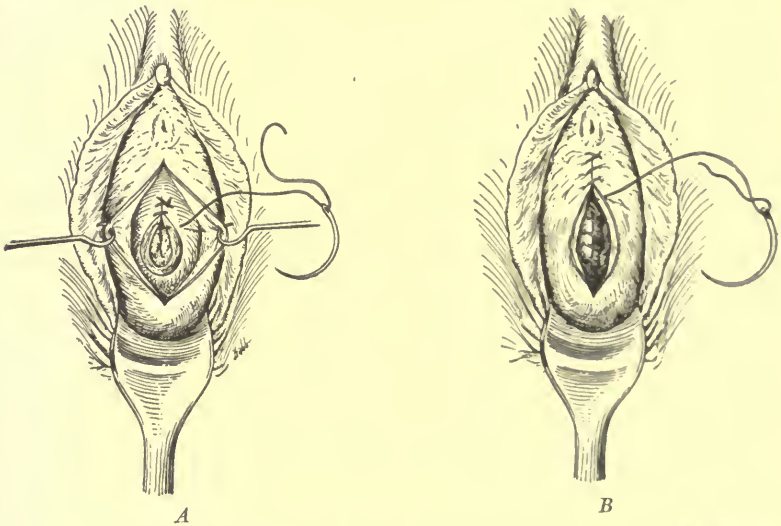
a few weeks and the more extensive ones are much reduced in size.

In the chronic cases an effort should be made to relieve coexisting cystitis before operative relief is attempted.

Vesicovaginal fistulae may be closed in the following way: The patient is placed in the Sims' or the lithotomy position; the cervix is delivered; the area around the adventitious opening to be denuded is outlined with the knife and removed with scissors; the tissues down to, but not including, the mucosa are excised. Interrupted chromic gut sutures (5 sutures to the inch) are introduced (not including the mucosa) ¼ inch from the edge of denuded area and tied. The possi-

bility of inclusion of the ureter in a suture must be borne in mind. The bladder is washed free from blood for the first few days. The vagina is packed for two days with iodoform gauze. Continuous vesical drainage by means of a rubber tube for four days, and subsequent catheterization every six hours for one week are essential. Vaginal douches are given after the third day.

For the relief of large vesicovaginal fistulae, *Mackenrodt's*[21] *operation* may be employed. The procedure consists in the complete mobilization of the base of the bladder from its vaginal and uterine attachments, and separate suture of the vesical and vaginal margins of the



A *B*

FIG. 973.— OPERATION FOR RELIEF OF LARGE VESICOVAGINAL FISTULA (Makenrodt).

A, Closure of fistula with suture line not including bladder mucosa; *B*, suture of the vaginal flaps.

fistula. In the larger defects the fundus of the uterus is used to occlude the opening in the bladder and the vaginal wall.

In the *closure of a vesico-uterine fistula* one of the four following methods may be used: (1) The anterior lip of the cervix is split to the fistula, which is denuded of scar tissue, and the entire wound is closed with silkworm gut sutures. (2) A transverse incision is made through the anterior vaginal fornix, the bladder is separated from the cervix across, and well above, the fistula; the vesical and uterine fistulae are closed separately, and the vaginal flap is reunited to the

cervix. (3) Mackenrodt's operation for vesicovaginal fistula (as stated above). (4) Through an abdominal incision the vesico-uterine fold of peritoneum is incised; the bladder and cervix are separated, exposing the vesical and uterine openings; the edges of these are refreshed and closed separately. The anterior peritoneal fold is sutured and the celiotomy wound is closed.

Vesico-uterovaginal fistulae may be closed by funnel shaped refreshment of the opening at the expense of the vaginal wall, and repair by simple suture. In large fistulae the anterior lip of the cervix may be utilized by denuding the edges of the fistula in this situation, together with excision of the edges of the opening in the vagina, and suturing the raw surfaces together in a transverse direction. If this fails, the abdomen may be opened and the repair made as described in (4) of the operations for the closure of vesico-uterine fistulae (see above).

The closure of a *ureterovesicovaginal fistula* may be accomplished by simply denuding the edges of the opening and turning the ureteral orifice into the bladder by means of interrupted chromic gut sutures.

For the relief of ureterovaginal fistula, Dudley[22] proceeds as follows:

Having located the ureteral orifice, a colpocystotomy is made so as to include, if possible, the ureteral opening in the incision, thus creating a ureterovesicovaginal fistula. To control bleeding the mucous membranes of the bladder and vagina bordering the fistula are united by sutures. A long, slender hemostat is passed through the vesicovaginal opening, one blade entering the ureter, the other, the bladder. The forceps is locked and by pressure necrosis (in three or four days) creates a ureterovesical opening at some distance from the vesicovaginal fistula which is closed later.

Colpocleisis is justified in cases of intractable vesicovaginal fistulae. The operation consists in removing a wide strip of tissue from the circumference of the vulvovaginal outlet. The raw surfaces are united by suture, thus converting the bladder and vagina into a common cavity.

Fecal fistulae are classified as rectovaginal, rectoperineal, and enterovaginal.

This class of lesion follows incomplete healing of pelvic lacerations, malignant, luetic and tuberculous ulcerations, and prolonged severe suppurative processes involving the pelvic organs.

The *diagnosis* is established by examination and, if doubt as to the exact nature of the vicarious communication exists, this may be cleared up by observing the behavior of injections of colored fluid.

The *operative treatment* is very similar to that described in connection with urinary fistulae.

VAGINITIS.— Vaginitis may be primary or secondary. *Primary vaginitis* occurs in connection with injuries to the vaginal mucosa. The secondary form results from extension of inflammatory processes from the vulva, uterus and urethra, and may be *acute* or *chronic*.

The *symptoms* are those common to all inflammations. Pain is aggravated by walking. The process is attended with a more or less abundant discharge, the character of which varies with that of the cause.

The *treatment* consists in rest of the inflamed part. The patient is put to bed and the vagina frequently (and gently) douched with soothing solutions. Strong antiseptics must be avoided. Of the various agents, mild zinc solutions (zinc sulphocarbolate, 1–1000) are as useful as any. Gonorrheal infection is taken up in the general part of this work.

Tuberculosis of the vagina may occur by extension from the uterus or vulva, or may develop primarily, the result of the introduction of tuberculous semen. The *clinical picture* of the disease is similar to that of tuberculosis of mucous membranes described elsewhere Part II, chap. xxiii). The *local treatment*, because of the accessibility of the process, is likely to be effective.

BENIGN GROWTHS OF THE VAGINA.— Cysts are usually of the inclusion class, being developed from remnants of Gartner's duct. The cysts may be single or multiple. They are usually small, but occasionally reach the size of a duck's egg. The content consists of a clear, yellow, viscid or watery fluid.

Vaginal cysts of this class rarely give rise to symptoms, unless they rupture. In this event the patient notes the presence of a persistent discharge. Their *extirpation* is readily accomplished.

Fibromyoma is perhaps the only benign tumor found in the vagina. The tumor is usually single, although multiple fibromyomata have been reported. Occasionally a pedunculated growth is twisted and the tumor undergoes necrosis. This is followed by spontaneous healing.

The tumor rarely gives rise to *symptoms* unless it grows large enough to interfere with coitus. This is generally regarded as sufficient to justify extirpation of the growth. Aside from this, however, the process may, by pressure, interfere with urination and defecation and even cause ulceration of the contiguous tissues.

Removal of the tumor is readily accomplished.

Malignant Growths of the Vagina.— Sarcoma of the vagina is very rare. It is usually situated in the anterior vaginal wall. In children the process makes its appearance in the form of a dull, red, circumscribed polypoid protuberance, while in the adult it is likely to present itself as a diffuse infiltrate, simulating carcinoma, or in the form of a rounded sessile mass which, sooner or later, ulcerates.

As a rule, painful micturition causes the patient to consult the surgeon, whose examination reveals the presence of the growth. Early *diagnosis* is possible only with the aid of the microscope.

The *treatment* consists in radical excision of the process. The *outlook* is unfavorable.

Fig. 974.— Carcinoma of Vagina.

Carcinoma is rarely primary in the vagina. It appears in the guise common to all carcinomata of mucous membranes (p. 1273). The inguinal glands are involved when the lesion is located in the lower one fourth of the vagina; higher than this, the deep pelvic nodes are invaded.

Intermittent, usually slight, bleeding attracts the attention of the patient. Later, a mucopurulent discharge appears. This finally becomes foul. Extension of the process is attended with pain which is often very severe. Ultimately the growth invades the bladder, or rectum, or both, frequently establishing fistulous communication with these organs.

The *diagnosis* is based on the appearance of the process and should be established by microscopical examination. All of the factors taken up in connection with carcinoma (Part VI, chap. xvii) must be given due consideration.

The *treatment* of course consists in early extirpation of the growth. Unfortunately the outlook is unfavorable at best. As yet, no case of healing has been reported. The employment of the palliative measures available in this class of cases makes life less unbearable; but they must be applied patiently.

Primary chorio-epithelioma is taken up elsewhere (p. 1285).

Surgery of the Internal Genitalia — Injuries to the Cervix and

Body of the Uterus.— Laceration of the cervix is the most frequent sequel of pregnancy. The laceration may be unilateral or bilateral. A stellate laceration is indicative of repeated labors.

The lesion is not attended with a *distinctive symptomatology.* It is uncovered in an examination in connection with the disturbances caused by endometritis and uterine subinvolution. Occasionally,

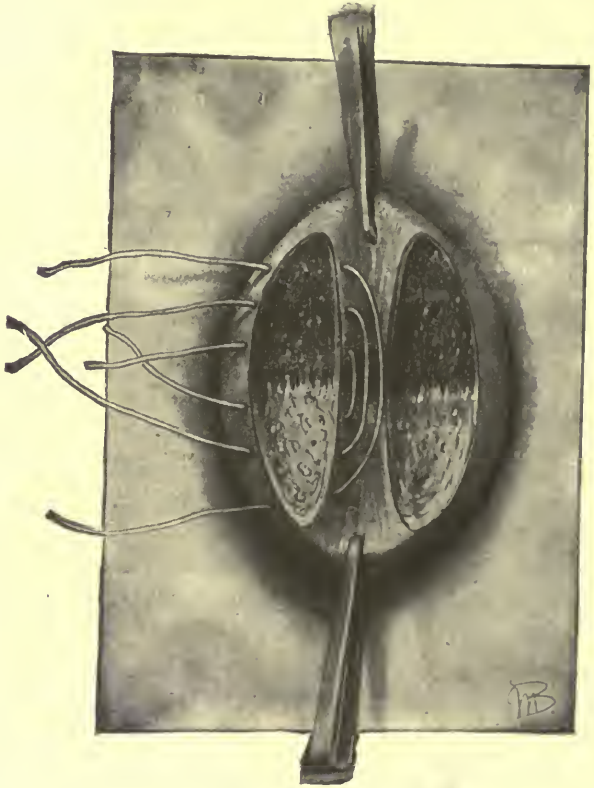


Fig. 975.— Trachelorraphy (Kelly).

bleeding from a granulating laceration attracts attention. The bearing that lacerations of the cervix have upon the subsequent development of malignant disease is not certain. However, a causative relationship cannot be excluded.

In the *treatment,* primary repair is not deemed necessary, unless the tear is responsible for severe bleeding.

In *trachelorrhaphy* (secondary repair) the cervix is delivered and held in place by means of tenacula, and the cicatrized coverings of the

cleft are denuded with the scalpel or scissors. Care must be taken to remove all scar tissue. Interrupted sutures are introduced in the manner shown in Fig. 975, and tied. The vagina is loosely packed with iodoform gauze for forty-eight hours, when it is removed and the vagina kept clean. Cleansing must be carried out with due attention to asepsis.

With regard to cases of laceration with marked *hypertrophy of the cervix,* Sturmdorf[13] says "Accepting the data from authoritative sources as a correct exposition of the facts, the obvious deduction is that since endometritis is the recognized pathological cause, trachelorrhaphy is an inadequate, and amputation of the cervix is an injurious, operation." Sturmdorf practices *tracheloplasty,* which he describes as follows: "(1) Outlining and free liberation of an ample cuff of mucosa from the vaginal sheath of the cervix. (2) Enucleation of the entire endocervical mucosa to the internal os, with preservation of the underlying muscularis. (3) Sutural inversion of the vaginal cuff into the denuded cervix."

Injuries of the uterus (nonpuerperal) are most often produced by instrumentation, of which the therapeutic are not by any means the least frequent. Unless the uterus is perforated and some of the abdominal contents are prolapsed, healing promptly follows careful mechanical cleansing and iodoform gauze tamponade.

In *prolapse of abdominal contents,* omentum, or gut (or both), the patient must be celiotomized. The prolapsed parts are drawn into the abdominal wound and, if necessary, repaired as in the manner taken up in connection with the Surgery of the Abdomen (Part XVI, chap. iii).

A discussion of rupture of the uterus during labor does not belong here.

Inflammatory Processes Involving the Uterus.— Inflammatory processes involving the uterus are classified as acute and chronic. Chronic inflammations are divided according to the location of the process, into *endocervicitis, endometritis,* and *metritis.* Endometritis is further subdivided into glandular and interstitial; the two may coexist.

Acute inflammation of the uterus most often follows childbirth, though it also occurs in the non-puerperal uterus. All the various forms of bacteria are responsible for the infection. Saprophytic and gonorrheal infections are the most common.

Saprophytic infection usually develops from three to ten days after

delivery and presents a picture of an acute infectious disease. The lochia are arrested, and examination reveals the presence of decomposing masses in the uterus.

In the *septic type of infection* the symptoms appear earlier but are much like those of the saprophytic form.

In both types of the disease the lower abdomen is tender. However, the temperature curve in the septic form is characteristic (see Fever, Part II, chap. v). Gonorrheal infection is differentiated by the history of the case and the microscopical examination of the vaginal discharge.

The *differential diagnosis* is important from the standpoint of the prognosis and the treatment. The *prognosis* is good in the saprophytic form; in the septic type the outcome depends upon the character of the infection. Pyogenic infection, if not fatal early in the disease, is attended with inflammation of the adnexa. This often terminates in a postuterine abscess or, which is unfortunately more common, the patient is left with chronic salpingitis, making necessary the sacrifice of these organs.

The *treatment* in the saprophytic form consists in emptying the uterus (dull curet). During the acute stage of septic and gonorrheal infection the patient is kept in bed and treated symptomatically. Postuterine abscesses are drained through the *cul de sac*. Other operative measures are contra-indicated during this stage of the process.

Endocervicitis occurs in connection with puerperal lacerations and injuries of various kinds, and is a part of a gonorrheal or septic genital infection. The process in the nullipara differs from that in the multipara. In the former the disease is attended with a small cell infiltration and dilatation of the capillaries; the squamous cells are infiltrated with leukocytes and become hypertrophied. The papillae become enlarged and are covered by a single layer of epithelium; their red color shimmers through the inflammatory exudate. Erosion of the mucosa is not uncommon. In multiparae, when the cervix is lacerated, the lips of the os are widely separated and are covered with irregular granulations. Eventually, the cervical glands are obstructed and become distended with accumulated secretion, so that the glands become cystic in character and are readily infected. The anatomical elements of the cervix gradually become greatly hypertrophied.

The *symptoms* of endocervicitis include more or less leukorrhea, pain in the back, and irregular menstruation. Sterility is the rule.

The *treatment* consists in excision of the cervical mucosa. See method of Sturmdorf, p. 2242.

Chronic endometritis may be divided into the glandular, the interstitial, and the mixed forms. The process is characterized by thickening of the mucosa, i. e., the glandular elements become elongated, tortuous, and dilated, and the cell elements enlarged, so that the proliferation resembles the structure of the decidua; at the same time the blood vessels are dilated and distended. In a certain number of the cases the endometrium is full of distended glands, which are responsible for a leukorrheal discharge; in others, the distended blood vessels dominate the situation, in which event the discharge is bloody; and in a third class of cases the glandular elements and blood vessels are absent and the membrane bears a resemblance to embryonic tissue. This is characterized by a mucopurulent discharge. Occasionally portions of the hyperplastic mucosa are exfoliated (*endometritis exfoliata*). Sometimes the membrane is elevated into polypoid masses consisting of glandular and vascular tissue (*polypoid endometritis*).

Endometritis is usually the outcome of infection following abortions and labor, and in the non-puerperal uterus is most often gonorrheal.

The cardinal *symptoms of endometritis* are hemorrhage and leukorrhea, the character of the latter being dependent upon the variety of the process. The discharge may be intermittent or constant. It is likely to emit a foul odor, exciting the suspicion of carcinoma of the body of the uterus. Prolonged retention of secretion may be responsible for salpingitis, oöphoritis, cellulitis, peritonitis, and pelvic abscess.

Chronic endometritis rarely heals except after curettement of the uterus.

Curettage is performed as follows: The patient is placed in the lithotomy position, and the vagina is cleansed. The cervix is drawn downward by means of a tenaculum and the canal dilated with bougies or a uterine dilator, and the depth and direction of the uterine cavity is ascertained with the sound. Retained placental tissue is removed with a sponge holder, and the endometrium is gently scraped with a dull curet. For the relief of chronic endometritis the mucosa is systematically scraped with the sharp curet. The uterine cavity is lavaged only to control bleeding, for which purpose hot saline solution is employed. The use of strong chemical agents is objectionable. The vagina is loosely tamponed with iodoform gauze, which is removed after forty-eight hours.

Chronic metritis is more or less associated with endometritis. The uterine muscularis is replaced by hypertrophied connective tissue, which later contracts (uterine fibrosis).

The *predisposing causes* of chronic metritis are retained placental tissue and blood clots, laceration of the cervix, and repeated abortions. These favor the invasion of bacterial excitants.

The *clinical picture* of chronic metritis is a complex one. The disturbances consist in indefinite pelvic pain, uterine colic, irritable bladder, constipation, dysmenorrhea, amenorrhea, and menorrhagia. In the early stages of the process, *examination* reveals a large, soft uterus; later, this organ becomes hard; it may become adherent to contiguous organs (bladder and rectum).

The *surgical treatment* in the early stages is limited to curettement. In the so-called fibroid stage persistent bleeding may make hysterectomy necessary. In the latter class of cases lues must be given consideration.

Hypertrophy of the cervix involves its vaginal and supravaginal portions. The latter form is associated with prolapse of the vagina. As a rule, the connective tissue and glandular elements of the cervix are involved in the process; the latter are often cystic.

Amputation of the cervix is indicated in both varieties of the disease. In the vaginal type the procedure is simple; in the supravaginal, it is necessary to dissect the vagina up to the peritoneal reflections. After the cervix is removed the mucosa of the stump is united to that of the vagina.

Tumors of the Uterus.— Of the *benign tumors* of the uterus *fibromata* are the most common. They are made up of fibrous tissue and muscle fiber (see Part VI, chap. xiii). Domination of fibrous tissue justifies the term *fibroma;* when muscle fibers predominate the term *myoma* is used, while a mixture of the two is covered by the term *fibromyoma.* As a rule, the entire uterus is hyperemic and enlarged and the endometrium is thickened. The growths are likely to undergo cystic degeneration; in old women they may be the seat of calcareous deposits.

Fibromyomata are *classified* in accord with their location as submucous, interstitial, and subperitoneal (Fig. 976).

Submucous fibromyomata (intramural, concentric) are situated beneath the mucosa and encroach upon the uterine cavity. As the tumor enlarges, it becomes more and more separated from the uterine wall, and finally is attached only by a pedicle, and then becomes a

polypus, or its base remains broad (sessile fibroid). The efforts that the uterus makes at expulsion of the tumor are responsible for a low grade of inflammation of the uterine structures, and if the growth is located at the fundus, its drag may cause partial or complete eversion of the uterus. In some instances the growth is incarcerated in the cervix, its pedicle is strangulated, and the mass is spontaneously amputated; in others, the tumor ulcerates, and this, together with the coincident endometritis, gives rise to a fetid discharge and to frequent bleeding.

Interstitial fibroids (mural or centric) develop in the wall of the uterus and may be circumscribed or diffuse. The former are likely to be multiple, while the diffuse are usually single and attain large dimensions. The lower segment of the uterus may be elongated in the form of a pedicle, which occasionally becomes twisted. The cavity of the uterus is also encroached upon by the process; this is attended with endometritis, while the entire organ is in a condition of chronic interstitial metritis.

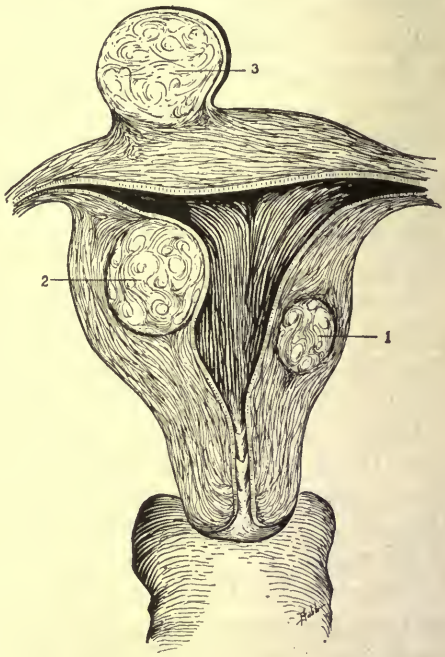


FIG. 976.— THE LOCATION OF UTERINE FIBROMYOMATA.

1, Interstitial; 2, submucous; 3, subserous fibromata.

Subperitoneal fibroids (extramural, eccentric, or subserous) are situated beneath the peritoneum, and as they increase in size, they grow on the surface of the uterus. Like the submucous, they may be broad based or pedunculated. They occasionally become adherent to the contiguous viscera, are separated from the uterus and receive their nutrition from the adjacent organ, thus becoming parasitic in nature. In some instances, the tumor grows out between the layers of the broad ligament, where it is likely to make pressure on the pelvic contents.

Hemorrhage is usually the *primary symptom* of fibromyomata. At

first, the menstrual flow is increased and prolonged, and is finally persistent. This symptom may be absent in cases of subperitoneal and interstitial lesions. Pain and a feeling of heaviness in the pelvis are usually complained of. As the tumors increase in size, the symptoms of pressure on the pelvic organs become manifest. These relate especially to the bladder and to the ureters (hydronephrosis) and rectum (constipation). When the intrapelvic growth becomes impacted, pressure on the nerves and vessels causes edema of the legs, and neuralgic pain in the various nerves of the sacral plexus. Submucous fibromyomata are likely to give rise to severe expulsive pains.

The *diagnosis* is based on the clinical history and the examination. It is not difficult, except in the early stages of the disease.

The surgical *treatment* aims at removal of the tumor or tumors. This is accomplished in various ways, depending upon the character of the growth.

Vaginal removal by torsion accomplishes the purpose when a pedunculated tumor presents in the cervix or vagina. The cervix is dilated or sectioned, the tumor is grasped with a tenaculum and its base snared off or divided with scissors. Occasionally a tumor of this sort may be removed piecemeal (morcellement).

Vaginal hysterectomy is employed in cases in which the tumor has not increased the size of the uterus too much. The vagina is separated from the cervix by a circular incision. The bladder is pushed upward until the peritoneum is reached. The posterior structures are opened to a similar extent. Both folds of the peritoneum are now opened, and the lower portion of the broad ligament on either side is clamped and divided between the clamps and the uterus. These sectors of the broad ligament are tied and the clamps removed. Next, the fundus of the uterus is drawn down through the anterior section, together with the tube and ovary of one side, and the broad ligament between the tube and ovary, and the uterus doubly deligated and sectioned between the ligatures from above. The opposite side is treated in a similar manner, thus extirpating the uterus. The stumps of the tubes and broad ligament are again transfixed and securely ligated. The sectioned layers of peritoneum are sutured and the upper ends of the vaginal opening closed. The operation has a restricted field of usefulness in this class of cases.

Transabdominal extirpation of fibromyomata is accomplished in two ways.

Myomectomy is a term applied to enucleation of the tumor from

the uterus. The method is simple and is perhaps permissible when the surgeon is certain that the lesion is single — a rare condition of affairs. As a rule, the pathology of the condition justifies *hysterec-*

FIG. 977.— CARCINOMA OF THE UTERUS (Kelly).

tomy. This is done in the manner described in connection with carcinoma of the uterus, except that the cervix is not removed and the tubes and ovaries are left *in situ.*

Malignant Growths of the Uterus.— Carcinoma of the uterus (Fig. 977) is the most common cancer in the female. Its frequent occurrence at the site of lacerations and chronic inflammatory processes is worthy of note. Histologically, the disease appears as a squamous or cylindrical cell lesion; the former is classed as an epithelioma, the latter as an adenocarcinoma.

Cancer of the cervix (like that of the tongue) begins either as a nodular growth of the glands embedded in its structure (adenocarcinoma) or as a diffuse infiltration of the cervical epithelium (epithelioma). Both varieties spread in all directions. The lymphatic involvement follows the course of the blood vessels between the layers of the broad ligament. When the process extends beyond the uterus, the latter becomes fixed and makes pressure upon the pelvic contents. Ulceration begins early and soon establishes communication with the bladder and the rectum. Extension of the disease to the general peritoneum occurs late.

Carcinoma of the body of the uterus presents the same histological picture as that of the cervix and soon infiltrates the entire organ. Ulceration also occurs early and lymphatic invasion is prompt. The latter appears in the pelvic lymph nodes and soon extends into those situated along the course of the iliac vessels and the aorta. The growth makes pressure on the various pelvic parts and organs, including the ureters. However, this is not so marked as in connection with fibromyomata.

Of the *symptoms,* the appearance of a moderate, bloody vaginal discharge in a woman past the climacteric deserves emphasis. This should arouse suspicion of the nature of the process and, as there are no pathognomonic symptoms, should lead to a careful examination. *Pain is a late symptom;* so is a fetid discharge. Bleeding (as such) also occurs late in the disease. The pain gradually becomes very severe and radiates in all directions. Preclimacteric bleeding manifests itself, at first as an increased flow; later, this is constant and is mixed with mucopurulent fluid. Cachexia develops early in the process. *Examination* early in the disease reveals a nodular thickening, or the cauliflower-like excrescences of the disease are palpable. The palpating finger often recognizes thickening and infiltration of the broad ligaments and determines that the uterus is fixed.

The *prognosis* depends upon the promptness with which operative measures of relief are employed. On the whole the outlook is not favorable.

Palliative measures of relief (p. 1283) are used in inoperable cases. Extension of the disease beyond the uterus itself makes a given case inoperable.

The operative measures, as in all cases of carcinoma, must be radical. This at once *excludes vaginal hysterectomy* from the problem.

Of the various methods of *hysterectomy,* the panhysterectomy of Wertheim is the operation of choice. The measure is executed as follows: The patient is placed in the Trendelenburg position, a low median celiotomy is made, and the gut dammed away from the pelvis with gauze packs. The ureters are exposed by opening the broad ligament at its base along the pelvic floor. They are then traced to the

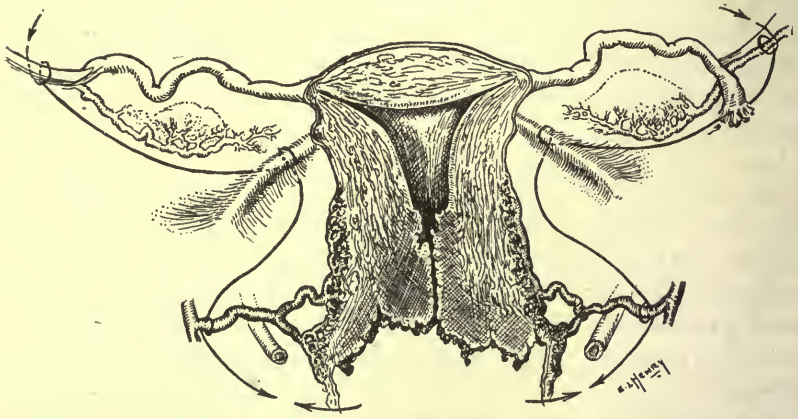


FIG. 978.— THE ESSENTIALS FOR ANY RADICAL OPERATION FOR CANCER OF THE CERVIX UTERI.

The excision of structures as here indicated must be carried out, whether the operation be abdominal or vaginal (Kelly).

base of the bladder, care being taken not to injure them. During the dissection the uterine arteries are exposed and divided between ligatures. The broad ligament is next divided as close to the pelvic wall as possible (Fig. 978), and its vessels are ligated. The peritoneum anterior to the uterus is incised, and the bladder liberated as far as the anterior fornix of the vagina. At the posterior aspect of the field, the rectum is freed in a similar manner. The uterus is now held in position by its vaginal attachments. The vagina immediately below the cervix is grasped with two right angled clamps and divided between them. If the conditions warrant, the posterior layer of the peritoneum is turned upward, exposing the iliac vessels and the glands

in this region, as far as the bifurcation of the aorta, are removed. The vagina is packed with iodoform gauze, and the peritoneum closed over it. The chief danger of the operation, aside from shock, lies in injury to the ureters, the bladder and the rectum. Of these the ureters are most often traumatized.

Sarcoma of the uterus is not frequent. It develops secondarily to fibroma in a certain number of cases. Sarcoma, like carcinoma, develops in the cervix or in the body of the uterus. It presents a clinical picture not unlike that of carcinoma, except that it shows a greater tendency to be tumorous in its growth and does not ulcerate as early as cancer. On the other hand, the process grows more rapidly than does carcinoma.

The *diagnosis* must be determined by the microscope. Chorionic epithelioma is taken up in Part VI, chap. xviii.

Uterine Displacements.— Of uterine displacements, Montgomery[23] says "The uterus is swung between the rectum and the bladder, occupying the axis of the pelvis at right angles to the axis of the vagina, its fundus reaching to a little above a line drawn from the promontory of the sacrum to the symphysis pubis, and the body slightly flexed forward on its cervix. Any continued deviation from this position must be considered a malposition."

The uterus may be displaced upward, forward, downward, backward, and laterally, producing ascent, descent, dislocations, versions, flexions, and inversions.

Ascent is produced by adherent growths, which become so large that they leave the pelvis, drawing the uterus with them. A collection of material in the pelvis may also cause the uterus to ascend. The condition is corrected by relief of the cause.

Descent, or prolapsus uteri, is one of the most frequent displacements, occuring both in the nulliparous and multiparous. Three degrees occur, (1) at a lower level in the pelvis; (2) when the os or a portion of the cervix protrudes from the vagina; (3) when the uterus, with, or without, the vagina, is situated outside the vulva (procidentia).

Prolapse of the uterus most frequently follows parturition when the pelvic floor is insufficient, i. e., laceration, overstretching of the levator ani muscles, loss of muscle tone, and increased abdominal pressure. The extra weight of the subinvoluted uterus favors prolapse of the organ.

The *disturbances* coincident with prolapse consist in dragging pelvic

pain and more or less interference with urinaton and defecation. As the prolapse descends, the protrusion of the organ becomes manifest. Persistence of the extrusion is attended with ulceration and inflammation of the prolapsed parts.

Operative measures of relief are preceded by correction of ulceration (tamponade). The method of procedure depends upon the cause of the prolapse. An elongated hypertrophied cervix is amputated. In supravaginal hypertrophy of the cervix the Wertheim-Watkins operation (p. 2234) may be employed. A lax or torn perineum is repaired as stated above (p. 2230). In severe cases the perineal operation is supplemented by ventral suspension of the uterus and shortening of the ligaments. In some cases it is also permissible to plicate the posterior fold of the peritoneum, thus drawing the cervix upward and backward. Hysterectomy is justified only when the uterus is diseased. Sturmdorf[13] claims that relief is afforded in even severe cases by (1) ample separation of the bladder base from the abnormal uterine and vaginal attachments acquired in the descent; (2) correction of the uterine malposition by vaginal reefing of the round ligaments; (3) reattachment of the bladder to the anterior surface of the uterus at the normal level (Goffe[24]); and (4) reconstruction of the musculofascial support of the pelvic floor.

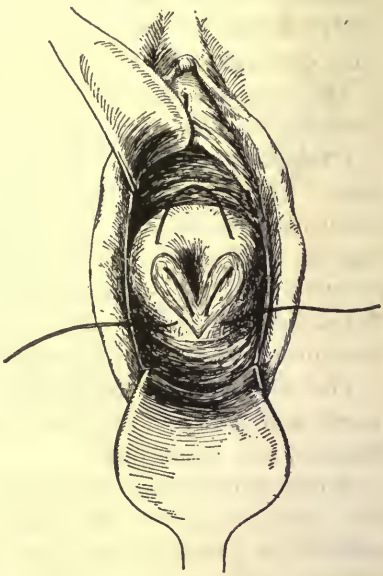


FIG. 979.— DUDLEY'S OPERATION FOR ANTEFLEXION.

Dislocation of the uterus may be anterior, posterior, or lateral; it is produced by inflammatory processes and possesses little surgical importance.

Uterine versions are the result of rotations of the uterus on a transverse (backward or forward), or on an anterior, axis (to either side).

In *anteversion* the fundus falls forward and, sometimes, downward. The malposition is produced by chronic metritis or other conditions which increase the weight of the organ. The concomitant symptoms are those of the causative condition.

In *anteflexion* the uterus is bent forward on its own axis. It is a frequent displacement in the virgin and nullipara. The infantile uterus is often anteflexed. The fully developed uterus is anteflexed as the result of inflammatory perimetritis.

An anteflexed uterus does not usually give rise to *disturbances*. On the other hand, sterility and dysmenorrhea are often charged to it by the gynecologist.

In cases of severe dysmenorrhea, Dudley splits the posterior lip of the cervix to its vaginal attachment, removes a section from either side and sews the edges of the cleft to the bottom of the incision (Fig. 979), thus making a new outlet for the uterus.

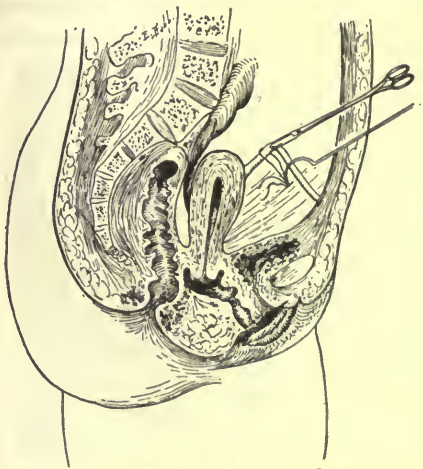


Fig. 980.— Montgomery's Operation for Retroflexion of the Uterus.

Retroversion of the uterus is most common in multipara and is usually a sequel of a lesion occurring during pregnancy.

The misplacement is not commonly attended with disturbances, but occasionally is responsible for dysmenorrhea and the widely distributed "pain in the back" of the female. The *treatment* is similar to that of retroflexion (see below).

Retroflexion of the uterus is frequently sequential to retroversion. The cervix is fixed and the fundus driven downward by the contraction of the healed inflammatory processes (adherent retroflexion).

The *disturbances* consist in painful menstruation and evidences of pressure upon the rectum.

In the *treatment* a movable reflexed uterus may, at times, be maintained in the normal position by means of a suitable pessary. When this fails, and when the uterus is adherent to the rectum or sigmoid (a common complication), operative measures of relief are justified. Many methods have been devised for the purpose. A résumé of most of these is presented by Montgomery,[23] who, like most writers, has discarded all the vaginal methods. Of the *abdominal methods*, which possess the advantage of permitting of mobilization of the uterus, that of Montgomery is described here in his own words:

"Through a median incision or, as I prefer, through a Pfannenstiel incision, each round ligament is caught about four centimeters from the uterus and secured by a temporary loop of catgut, the ends of which are threaded into the eye of a Deschamp needle. Through an opening made in the anterior leaflet of the corresponding broad ligament, the needle is carried outward between the two layers of the ligament until it reaches the point where the peritoneum is reflected onto the abdominal wall, where it is carried through and unthreaded, and the ends of the ligature secured by forceps (Fig. 981). The same steps are followed on the opposite side. The ligature is then put on the stretch, a pair of sharp pointed scissors inserted alongside it, closed, and the blades slightly separated, after which a loop of the ligature is easily drawn through and is secured to the outer surface of the aponeurosis by chromic catgut sutures. The loops are drawn through sufficiently to bring the fundus of the uterus well forward."

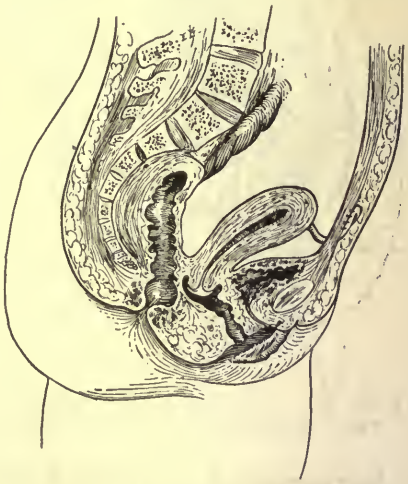


FIG. 981.— MONTGOMERY'S OPERATION COMPLETED.

Inversion of the uterus is divided into three stages, (1) intrauterine, (2) intravaginal, and (3) extravaginal.

The lesion is the outcome of a relaxed condition of the uterine walls precipitated by traction on the cord in forcible efforts to deliver the placenta, or by an effort on part of the uterus to expel a fibroid tumor.

Acute inversion may be attended with profound shock and severe pain. In the less precipitate cases, the diagnosis is made upon vaginal examination.

The *treatment* in acute cases consists in manual reduction of the extrusion. If this fails, splitting the posterior cervix may be resorted to. In persistent cases hysterectomy is justified. In instances in which the inversion shows a tendency to recur, it is permissible to fix the uterus in its normal position (see prolapse).

Surgery of the Fallopian Tubes.— Inflammation of the fallopian tubes (salpingitis) is of great surgical importance in connection with

the differential diagnosis in acute abdominal conditions. The disease is most often due to *gonorrheal* infection, but also develops as the outcome of *pyogenic* invasion and is, occasionally, *tuberculous* in character. In accord with the nature of the provocative agent, salpingitis is acute, subacute, and chronic.

The *significance* of salpingitis, like that of appendicitis, lies, as far as life is concerned, in the attendant invasion of the peritoneum. In addition to this, the disease is responsible for sterility.

Pyogenic salpingitis follows a course similar to this class of infection in all organs covered by peritoneum, its healing being at-

FIG. 982.— PYOSALPINX (Kelly).

tended with coalescene and contractural deformation of contiguous parts and organs, and more or less interferenee with their funetion. The formation of collections of inflammatory exudate in certain seetors of the tubes constitutes foei of chronic inflammation whieh give rise to more or less frequent renewed attacks of localized peritonitis. Occasionally a collection of this sort ruptures and is followed by fatal general peritonitis. In another elass of cases the focus (pyosalpinx) ruptures into the uterus, bladder or rectum. However, this is rare.

Tuberculous salpingitis begins in the mucosa of the tube by extension from the uterus. It may be hematogenous in origin, or the tube may become involved as a part of a tuberculous peritonitis. The last is the rule. Spontaneous healing may be followed by cicatricial obliteration of the tubes, or these may undergo caseation or calcification.

The *symptomatology* of salpingitis is essentially that of pelvic peri-

tonitis. The disturbances are most manifest in the lower segment of the abdomen. Unilateral (right) salpingitis is readily mistaken for appendicitis. The *differential diagnosis* is made by careful consideration of the history of the case, the local findings, the subjective symptoms, and the blood picture. As the case progresses, the process either merges into a chronic salpingitis or develops into a pelvic abscess. After the inflammation has subsided, the patient is subject to more or less frequent exacerbations of pelvic symptoms, i. e., pain, tenderness, disturbances of micturition and defecation, and menstrual irregularities. In these cases vaginal examination reveals the presence of the pathological tubes.

The *diagnosis* is difficult only in the acute stage of a primary attack. Recognition of the condition of affairs has an important bearing upon the treatment.

The almost universal *treatment* of acute salpingitis consists in placing the patient in bed, using hot vaginal douches, applying the ice bag to the abdomen, and administering opiates. The rationale of this lies in the assumption, which is usually correct, that the inflammatory process will subside and that operative efforts at relief simply spread the infection. While this is contrary to the general rule, the removal of parts and organs the infection of which involves danger of fatal peritonitis, it is nevertheless true that a fatal outcome ensues more often when acutely infected fallopian tubes are removed than when subsidence of the active process is awaited. That there are, however, cases of virulent pyogenic infection of the tubes, which are subject to the general rule of establishing an opportunity for the egress of inflammatory exudates would seem to be equally true. It is certain that the subsidence of gonorrheal salpingitis should be awaited; in pyogenic infection the surgeon must be guided by the special indications deduced from the clinical picture presented by a given case. It is of course patent that operative measures which make possible the leaving *in situ* a part or an entire ovary, are most likely to be successful when the acute inflammatory process has subsided. On the other hand, there seems to be no reason why a case of general peritonitis originating from the uterine adnexa should not be subject to the same rules of surgical treatment as that developing in connection with the other abdominal organs (gallbladder, ileum, colon, appendix, etc.). Consideration of the problem from the standpoint of general surgical principles may be properly suggested to the gynecologist.

Hydrosalpinx.— Hydrosalpinx consists in distention of the fallo-

pian tubes with mucoid secretion, following a chronic inflammatory process. The tube undergoes all sorts of distortions and is irregularly protruberant, in some instances representing a cystic enlargement of considerable size. Tubes of this sort present no distinctive clinical picture and are usually not recognized until exposed in connection with celiotomy employed for the relief of the causative chronic inflammatory process. No doubt the coincident atresia is often responsible for tubal pregnancy.

Ectopic Gestation.— Ectopic gestation is a term used to designate the development of a fecundated ovum outside the uterine cavity. It is divided into *tubal, ovarian* and *abdominal.*

The *cause* of ectopic pregnancy is not certain, but it is probably due to anatomical (congenital) or inflammatory atresia of the lumen of the fallopian tubes.

Tubal pregnancy is classified as (1) *interstitial* or tubo-uterine; (2) *true tubal;* and (3) *tubo-ovarian,* or infundibular.

As gestation progresses, the muscularis of the tube stretches and becomes hypertrophied; the mucosa is thickened and takes on the character of a decidual membrane. Finally, the muscular fibers disappear and are replaced by fibrous tissue, which ultimately atrophies at the site of the placenta and ruptures, thus liberating the ovum, which escapes between the layers of the broad ligament or into the abdominal cavity. From now on, the ovum either continues its development or dies. In the former instance the placenta establishes parasitic attachment to other structures. Concomitantly with these processes, the uterus enlarges and becomes lined with a decidual membrane, but this ultimately undergoes regressive changes.

The *clinical picture* of ectopic pregnancy is an exceedingly varied one. It may develop in a nullipara (congenital tubal atresia) or follow inflammatory lesions of the tubes. In most instances, the condition is preceded by a more or less protracted period of sterility. This is followed by indefinite pelvic pain, suggesting a threatened abortion, and a blood stained vaginal discharge makes its appearance. The early signs of pregnancy may be present, but usually are absent. Vaginal examination is likely to reveal a well defined movable mass on either side of the uterus or in the *cul de sac.*

Intraperitoneal rupture of the tube occurs during the period from the sixth to the eighth week. This is usually attended with severe hemorrhage; on the other hand, bleeding may be slight and may recur at frequent intervals, so that gradually a hematocele is developed.

In some instances the expelled ovum continues to grow in the abdomen, forming parasitic attachments to the contiguous organs. As a rule, however, rupture of a tubal pregnancy is attended with severe *shock and the classic picture of internal hemorrhage.* The abdomen is distended and tender.

Extraperitoneal rupture does not usually occur until from the seventh to the twelfth week of pregnancy. Vaginal examination reveals the presence of a large tense or doughy pelvic tumor; the ovum promptly dies. Subsequently the effused blood may be absorbed or become infected, and a pelvic abscess may develop.

Occasionally an ectopic pregnancy goes on to the fourth month, presenting a picture not unlike that of uterine gestation. In this event the fetus may become encapsulated and remain in its abnormal environment (with little disturbance) for a long time, and finally perforate into a contiguous hollow organ.

All cases of ectopic pregnancy should be celiotomized as soon as the diagnosis is made. The tube is promptly deligated and ablated. If the fetus lies free in the abdomen, it is removed, together with the effused blood — not a difficult maneuver. A hematocele or a complicating pelvic abscess is drained through the *cul de sac.*

Surgery of the Ovary.— Inflammatory processes of the ovary are divided into those occurring in the substance of the ovary (*oöphritis*) and those involving the tissues surrounding it (*peri-oöphoritis*).

Oöphoritis may be acute or chronic, gonorrheal or pyogenous, and is characterized by all the signs and symptoms of inflammation. It usually occurs in connection with salpingitis, but occasionally stands alone, the outcome of hematogenous infection. Ovarian abscess, except when coincident with salpingitis, is very rare.

Peri-oöphoritis is attended with thickening of the tunica albuginea, which interferes with rupture of the graafian follicles and may thus be responsible for sterility. Prolongation of the process is responsible for cystic degeneration of the ovary, or the ovary becomes adherent to the tube or uterus (often in the *cul de sac*).

Neither oöphoritis nor peri-oöphoritis produce a distinct *clinical picture.* In advanced cases the enlarged cystic ovary, which gives rise to indefinite pelvic pain, may be palpated.

The *treatment* is similar to that of salpingitis. Oöphorectomy is an objectionable operation and is rarely justified, except when the ovary is involved in a suppurative process.

Ovarian Tumors.— Growths of the ovaries may be divided into cystic and solid.

Cysts of the ovary show a greater tendency to become malignant than those situated elsewhere in the body. They are divisible into the simple proliferating (true ovarian), cysts of the corpus luteum, and

Fig. 983.— Ovarian Cyst (Symmer's Bellevue Hospital case).

dermoids. *Solid tumors* are usually fibromata, sarcomata, carcinomata, or endotheliomata.

True ovarian cysts (proliferating cysts, cynstadenomata) are frequently multilocular and usually involve both ovaries. They originate in the ovarian epithelium of the graafian follicles. Large cysts are likely to become unilocular; in this event, small cysts project into the interior of the larger. The contents of the cysts consist of a tenacious, straw colored fluid. However, if bleeding occurs, the fluid is dark in color. In size the cysts vary from that of a lemon to one occupying the entire abdomen, and may embarrass respiration. The cyst is

covered with peritoneum; the broad ligament and tube is stretched over it and forms its pedicle.

Parovarian cysts may be mentioned at this time; they possess the same characteristics as the ovarian, but are likely to contain papillomatous outgrowths, which perforate the wall of the cyst and become engrafted onto more or less of the peritoneum. They bear some resemblance to malignant cysts of the mammary glands.

Cysts of the corpus luteum are usually multiple and may attain the size of a lemon. They contain a dark, tenacious fluid.

A *dermoid cyst* is usually unilocular and grows very slowly (see Part VII, chap. i).

Of the solid tumors:

Fibromata attain only moderate size. They are very rare.

Sarcoma of the ovary is also rare. It is likely to be cystic in character and to grow very rapidly.

Carcinoma of the ovary is not so rare. Twenty per cent of ovarian cysts exhibit malignant changes.

The *symptoms* of ovarian tumors are not clearly defined until evidences of pressure develop. The latter relate especially to the bladder and the rectum. As the ovary is a loosely attached organ, these disturbances may never be an important factor, unless the growth becomes malignant. As a rule, enlargement of the abdomen attracts attention and this leads to an examination, which reveals the nature of the condition.

Inspection reveals symmetrical enlargement of the abdomen; the flanks do not bulge. *Palpation* outlines the growth and elicits fluctuation. Bimanual examination shows that the growth is more or less movable, and that the uterus is dragged upward out of the pelvis. The *percussion note* is dull and does not vary with the position of the patient.

Inflammation of an ovarian growth occurs in connection with infection of the uterine adnexa. This may lead to a confusing clinical picture.

Torsion occurs in ovarian growths with long slender pedicles. The result of torsion varies with the degree of the axial displacement, and finally results in strangulation and gangrene.

Torsion is attended with sudden severe pain and rapid increase in the size of the cyst.

Rupture of an ovarian cyst may be spontaneous or occur as the result of trauma.

The *diagnosis* of rupture is possible only when the cyst has been previously recognized and its outline suddenly disappears. In other instances, the presence of shock and the evidences of a severe intra-abdominal lesion justifies celiotomy, which leads to discovery of the condition.

In the examination of tumors of the abdomen, the establishment of their connection with the ovary is not by any means always easy. The surgeon must take into account a distended bladder, a pregnant uterus, and ascites.

Ovarian tumors should be removed. This may, occasionally, be accomplished through the vagina. The method is unsafe and succeeds only under extraordinary conditions. The problem is best approached through the abdomen. Large cysts should be emptied (trocar and cannula) before delivery. Adhesions often complicate the technic; they must be carefully separated. Finally, the ovarian or the broad ligament is deligated and the tumor removed. When both ovaries are the seat of tumors, an effort should be made to leave a portion of ovarian tissue *in situ*. Preservation of the function of the ovary as an internal secreting organ is very important. This is equally true with regard to salpingectomy, made necessary in connection with inflammatory processes in this situation. The *conservative surgery of the tubes and ovaries* is made the subject of an extensive presentation by Kelly,[25] who also submits the bibliography in this connection.

The *technic of salpingectomy and salpingo-oöphorectomy* should aim at the removal of the pathological process only. For the purpose the patient is celiotomized, preferably in the Trendelenburg position, and the offending part excised in accord with general surgical principles. It would seem permissible to add that measures addressed to the tubes, directed toward maintenance of their lumen, are not likely to be successful and that the surgeon should aim at preservation of the tissues concerned in furnishing an internal secretion, i. e., that of the ovaries.

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PART XIX

THE SURGERY OF THE GENITO-URINARY SYSTEM

CHAPTER I

THE SURGERY OF THE KIDNEYS, URETERS, AND SUPRARENAL GLANDS

Anatomical Considerations.— As one is justified in assuming that the reader is familiar with the anatomy of the kidney and its superimposed tissues, only a few of the factors in this connection, those that possess surgical importance, are taken up here. These consist in the structures enmantling the kidneys, their blood vessels, and the topographical relationship the kidneys and ureters bear to the organs contiguous to them. The kidney parenchyma is closely covered by a thin, translucent layer of firm fibrous tissue — the *tunica fibrosa, propria, or albuginea.* Normally, this may be readily separated from the kidney; at the hilum, it continues on over it and extends into the substance of the organ, forming the outer layer of the papillae and calices. The capsule also sends a prolongation over the blood vessels and nerves as far as the aorta and vena cava, and a second one (much thicker) is insinuated between the renal vessels and the ureters, finally merging with the sheath of the aorta and the lumbar fascia. The latter process is called the ligamentum suspensorium renis (Kümmel[1]).

The proper capsule of the kidney is connected by numerous delicate prolongations with its *fatty capsule,* which is thickened at the outer border, the lower pole, the hilum, and the posterior aspect of the organ.

A readily discernible thickening of the connective tissue situated at the posterior aspect of the kidney is called the *retrorenal fascia* (Glautenay and Gosset[2]). However, the anterior portion of the kidney also possesses a more or less fully developed fascial structure called the *fascia prerenalis;* it is always present on the left side where it is connected with the retroperitoneal connective tissue space of the mesocolon (descending) and extends upward to the pancreas and is lost in the sheaths of the vessels located in front of the vertebral column.

The two layers of the fascia coalesce at the convex border and the

upper pole of the kidney; at the median border they remain separated; the posterior layer is lost in the region of the spinal column; the anterior extends to the corresponding layer on the other side of the body. At the lower pole the fascia also remains separated and merges with the pelvic fascia. In this way the kidney is surrounded by a third layer, which is called the *renal fascia*. In front, it is closely attached to the peritoneum; behind, it is loosely connected with the sheaths of the psoas and quadratus muscles. Outside of this third layer the kidney is surrounded by a stratum of subperitoneal fatty tissue. From this it will be seen that the kidney is not very securely maintained in its normal position.

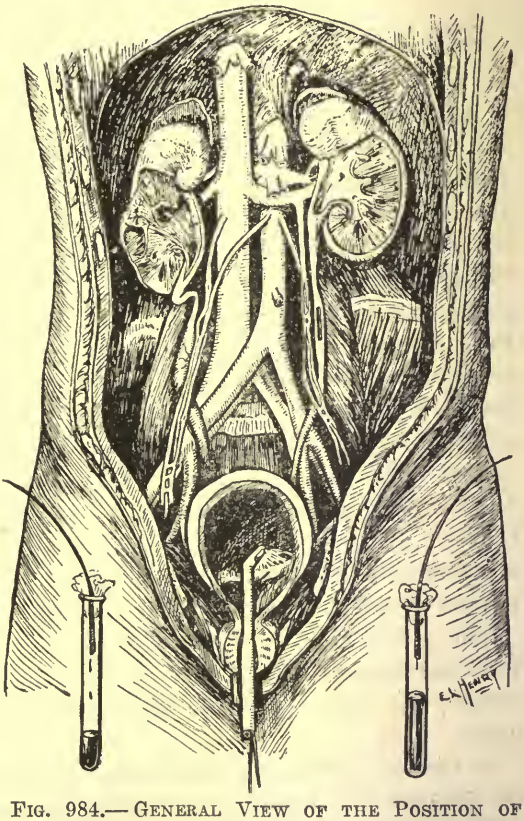

FIG. 984.—GENERAL VIEW OF THE POSITION OF THE KIDNEYS, URETERS, AND VESSELS.

The right kidney is tuberculous; the left is normal. The ureters are catheterized.

The kidneys are located in the lumbar region on either side of the spinal column and occupy the upper third of the lumbar fossa. Normally, they extend from the upper border of the twelfth dorsal to the lower edge of the second or the middle third of the third lumbar vertebra. The right kidney usually lies a finger's breadth lower than the left.

The posterior surface of the kidney rests with its median edge on the quadratus lumborum muscle, and its lateral edge rests on the transversalis muscle. At this point it is in contact with the thinnest portion of the posterior abdominal wall and is covered only by the edge of the sacrolumbalis muscle, the lumbdorsal fascia and the transver-

salis muscle. The upper pole of the kidney reaches to the region of the pleural cavity. The lower aspect of the pleural cavity is, therefore, of great surgical importance.

Normally the pleura is reflected upon itself on a line drawn from the lower edge of the body of the twelfth dorsal vertebra, which crosses the eleventh rib 3 to 4 cm. behind its attachment, and ends at the cartilage of the same rib. According to Luschka,[3] the lowermost reflection of the pleura is located 4½ cm. from the outer end of the cartilage of the eleventh rib. According to Recamier,[4] the pleura descends at the side of the vertebral column to a point 1 or 1.5 cm. below the twelfth rib, then follows an oblique downward and outward course, crosses the twelfth rib and the last intercostal space, and, still following its downward obliquity, crosses the eleventh rib at its lowermost point, i. e., from 10 to 12 cm. from the median line. This means that the outer end of the twelfth rib, which extends over the sacrolumbalis muscle, is not covered by the pleura. As nephrectomy involves manipulation close to, and at times resection of, the twelfth rib, the relationship that it bears to the pleura must be taken into account.

The kidneys are located behind the peritoneum, which skirts their anterior surfaces. The relationship of the peritoneum to the right kidney differs from that to the left, however. On the right side the peritoneum dips down from the liver and covers the kidney where it lies in contact with this organ, leaving the lower third free. On the left side, the peritoneum covers the kidney only where the latter is in contact with the stomach — a small area.

The upper two thirds of the *right kidney* is in contact with the liver, the lower one third lies behind the hepatic flexure of the colon, its medial border in close relation with the vertical portion of the duodenum, and connected with it by a dense fold of peritoneum, which also fixes the duodenum to the vertebral column. For this reason, perinephritic abscesses may rupture into the colon or duodenum. The suprarenal gland is wedged between the liver and the upper pole of the kidney. The lower pole lies behind the colon.

The upper third of the *left kidney* is in contact with the stomach, the middle with the pancreas, and the lower with the posterior peritoneum. The left suprarenal gland lies above the hilum. The upper, lateral edge of the left kidney touches the spleen; the lower, the colon.

The posterior surfaces of the colon, in contact with the anterior portion of the kidneys, are extraperitoneal.

The *ureter,* immediately at its upper portion, comes in contact with the psoas major muscle, which it follows downward and inward (behind the peritoneum) into the pelvis and on to the posterior wall or base of the bladder, passing through the latter in an oblique direction. At the middle of the psoas muscle (or slightly lower) the ureter passes behind the spermatic artery. Lower down, the ureter crosses the common iliac vessels close to their bifurcation. The right ureter lies behind the ileum, the left behind the sigmoid. The *lumen of the ureter* varies at different portions. It is exceedingly elastic. Three normal points of constriction are found as follows: (1) opposite the lower pole of the kidney; (2) where it crosses the iliac vessels; (3) as it enters the bladder.

The kidney is supplied by the *renal artery,* which originates from the aorta and divides before it enters the hilus; it lies, together with the *vein,* in front of the pelvis.

Methods of Examination.— The hidden position of the kidney renders inspection practically impossible, and the organ is also almost inaccessible to palpation. However, both of these are of some value in this connection.

Inspection reveals gross swelling, such as occurs in connection with suppuration, retention of excretion, tumors, effusions of blood; and, at times, in thin persons, displacement of the kidney may be visualized.

Palpation possesses distinct value. The maneuver is executed in the following manner: The patient is placed in the semirecumbent position; this causes the kidney to sink downward and relaxes the abdominal muscles; one hand is placed behind and below the twelfth rib, and the other rests on the abdomen. In this way it is possible to palpate a malplaced or enlarged kidney, especially during moderate inspiration. Deep inspiration makes the abdominal muscles tense. Both sides must be palpated in all instances.

It is important to *determine the relationship of the colon to a tumor in the kidney region.* If the colon is distended with gas (artificially, per rectum), the presence of a tympanitic percussion note over the tumor is of considerable diagnostic value. This does not, however, exclude enlargement of the right lobe of the liver, as the distended colon is often postured in front of it. Occasionally, a distended gall-bladder causes confusion in this connection. On the left side, enlargement of the kidney must be differentiated from that of the spleen. The peculiar conformation of the latter should make the distinction.

Röntgenography is, of course, of diagnostic value; it reproduces

the *kidney shadow,* makes possible the recognition of *stones,* and is particularly valuable when employed in connection with pyelography, which visualizes the outline of the form of the pelvis and calices of the kidney and the conformation of the ureters. Although the technic of Röntgenography is much improved, it may not yet be regarded as conclusively indicative in a negative sense. On the other hand, the recognition of the conformation of the kidney has been materially aided by obtaining a Röntgenographic negative of the abdomen with the latter filled with oxygen. The technic of the procedure is presented by Stewart.[5]

As in all diseases of the urinary passages, recognition of surgical lesions of the kidney is materially aided by *examination of the urine.* A number of these are attended by parenchymatous and interstitial inflammatory conditions, which are responsible for the presence of albumin and casts. Others are responsible for the admixture of blood and pus, the *renal origin* of which is established by various means.

Hematuria varies widely with respect to the quantity and character of blood voided. At times the blood is expelled in long clots, which correspond in form to the ureters; their propulsion into the bladder is attended with more or less renal colic. At other times, and more commonly, the urine is mixed with blood and contains blood cylinders. The presence of microcytes, i. e., disintegrated red corpuscles, is regarded as very indicative of renal bleeding.

The *presence of pus in the urine* is less indicative. Its renal origin is probable when pus is associated with granular, epithelial, and pus casts. However, the absence of casts does not exclude the existence of a suppurating kidney.

The determination of the renal origin of pus is not sufficient for diagnostic purposes. It is necessary to ascertain from which kidney the pathological urine is excreted. Extirpation of a diseased kidney is justified only when the other is shown to be normal.

The history of the development of the methods of obtaining the urine from each kidney, interesting and instructive as it is, cannot be reproduced here. For our purpose it must suffice to say that satisfactory methods were not possible until the advent of electric illumination of the bladder, which also made the ureteral openings visible and rendered them accessible to instrumentation (Nitze,[6] Fenwick,[7] Caspar,[8] Albarran,[9] Luys[10]).

Cystoscopy.— Visualizing the interior of the bladder is of diagnostic value as far as kidney lesions are concerned, inasmuch as the con-

dition of the former bears a relation to the latter, and because of the information obtainable as the result of observing the ureteral openings. If abundant clear urine is expelled from one ureter, and the other ejaculates sparse quantities of thick, purulent or bloody urine, the problem is solved. In other instances, the presence of ulcerations in the region of the ureteral openings gives valuable information. The value of negative findings is shown by the fact that, when repeated examinations reveal only a single ureteral opening, it is justifiable to assume that the patient possesses only one kidney.

Ureterocystoscopy.— However, the most important progress in urological diagnosis is represented by the development of *ureterocystoscopy*, which permits of the introduction into the ureters of catheters through which the urine of either kidney is obtained. Of the various instruments devised for the purpose (too numerous to describe here) that of Bierhoff[11] shown in Fig. 985 is indicative of the possibilities in this connection. The instrument permits of simultaneous catheterization of both ureters and lavage of the bladder at will. The optical portion,

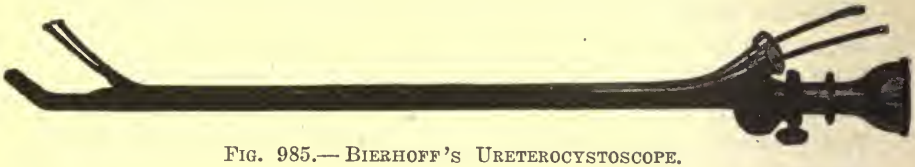

FIG. 985.— BIERHOFF'S URETEROCYSTOSCOPE.

to which the lamp is fixed, is movable, and avoids crossing of the ureters when the instrument is withdrawn. An elaborate presentation of various forms of ureterocystoscopes is submitted by Luys.[10]

Introduction of the instrument into the bladder is accomplished in the manner stated in connection with searching of the bladder. Once the bladder is entered, the patient does not experience much discomfort. The silk catheters are placed in the instrument before it is introduced into the bladder, which should contain from 150 to 200 c.cm. of clear fluid. Narcosis is rarely necessary. However, the injection of a one per cent novocain solution is desirable.

After the ureteral orifice is located the catheter is advanced into the field of vision and carefully guided into the former. The maneuver requires some practice. When the catheter is engaged in the ureter it appears to the examining eye as shown in Fig. 986. At this time the catheter guide is drawn slightly backward; this permits of advancing the catheter with less angulation. The mandarin is withdrawn and the catheter is left *in situ*. If urine flows in a stream, the end of

the catheter lies in the pelvis of the kidney or has been accidentally withdrawn into the bladder. When the catheter is in the ureter, urine flows intermittently. The urine is caught in receptacles in the manner shown in Fig. 984.

Despite the good services rendered by the ureterocystoscope in the segregation of the urine of each kidney, efforts have been made to accomplish the purpose by creating a temporary division of the bladder, into each of which divisions the urine of the respective kidney is collected and later withdrawn. A number of methods have been devised for the purpose. Of these the separator of Chatelin, classic in the literature, may be regarded as exemplifying the principles

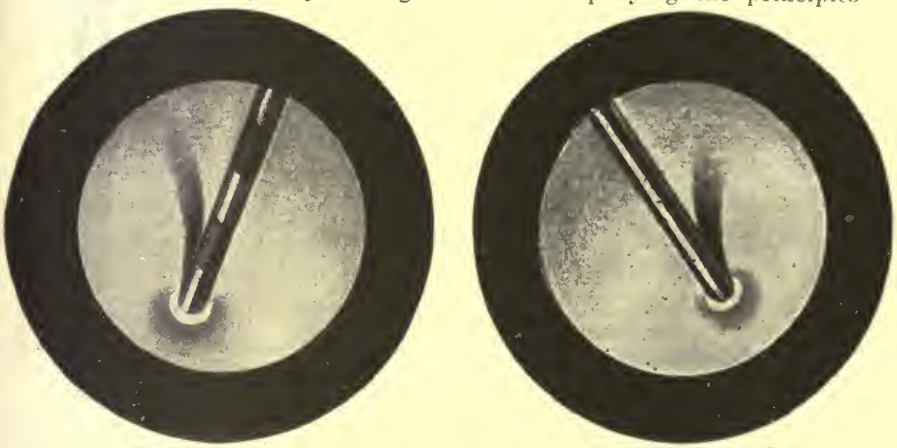

FIG. 986.—CYSTOSCOPIC VIEW OF A CATHETER ENGAGED IN THE URETERAL OPENING.

involved. It is permissible to state that this and similar methods are rarely employed at this time.

Catheterization of the ureters through the *direct cystoscope* is employed in cases of contracted bladder. The method was perfected in its application to the female bladder by Kelly,[12] and by Luys[10] in connection with the male. While the method is presented in a seemingly convincing manner by Luys (translated by Wolbarst) it is not practiced extensively by American surgeons at this time. However, there would seem to be no doubt that the measure possesses certain advantages with regard to the diagnosis of doubtful intravesical conditions, especially as it obviates the deformation of the picture coincident to indirect cystoscopy. The instrument devised by Luys[10] is very simple (Fig. 987).

Determination of the Kidney Function.— The successful outcome of

surgical attack upon the kidney is much dependent upon whether the sister organ is capable of sufficient function to meet the demands. It is necessary to determine what the quantitative output of one or both kidneys is in a given period of time. In determining the kidney function, the tests are quantitative and relate primarily to recognition of the total capacity of the kidneys; secondly, to the capacity of a single kidney. Cryoscopy of the blood measures the total excretory function of the kidneys by indicating the degree of retention of end products. The additional methods are concerned in measuring the function of a single kidney. The simplest methods are the following:

1. Estimation of the quantity of urea excreted.
2. The phlorizin method.
3. Chromocystoscopy.
4. Phenolsulphonphthalein method.
5. Estimation of the density of the solution by its electrical conductivity.
6. Experimental polyuria.
7. Cryoscopy of the urine.
8. Cryoscopy of the blood.

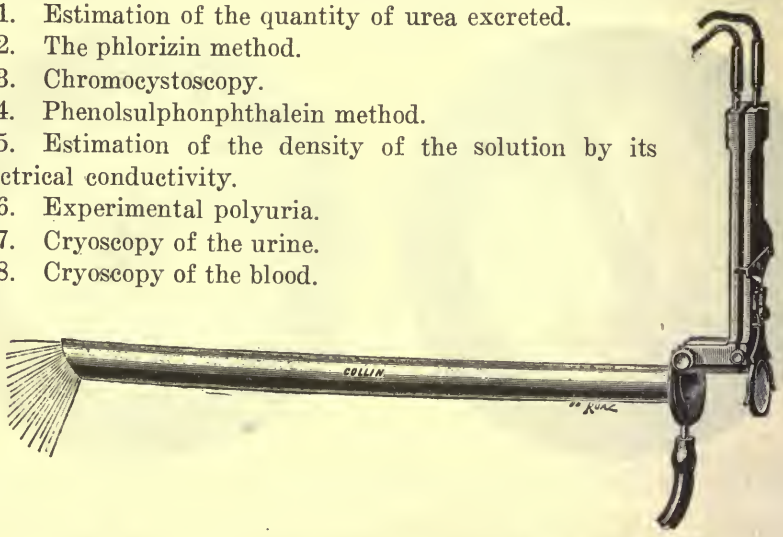

FIG. 987.— LUYS' DIRECT VISION CYSTOSCOPE FOR THE MALE.

The female instrument is somewhat shorter (Luys).

None of these tests is determining. Several must carry out an analytical accord.

1. The diagnostic significance of the urea content of the urine of one kidney is regarded as indicative of the functional capacity of the organ as compared to that of the other. For the purpose, the *Esbach method* is sufficiently accurate.

2. The *phlorizin method* standardizes the functional activity of the kidney on the basis of a *renal glycosuria* (Richter[18]). One to one and one half mg. of a one to two per cent phlorizin solution is injected hypodermically; sugar is excreted by the healthy kidney in from

twenty to thirty minutes; this is markedly delayed when the kidney is diseased.

3. *Chromoscopy* is based on the elimination of colored substances which, when introduced into the body, are eliminated in the urine. Of these *indigocarmin* is the most satisfactory (Völker and Joseph[14]). Twenty c.cm. of a 0.4 per cent solution in 0.6 per cent sodium chlorid is injected into the gluteal region, and may (normally) be observed in the urine expelled from the ureter in twenty minutes in the form of a blue fountain. The method may be used for the purpose of locating the ureteral opening in cases of ulcerative cystitis, etc., and to establish the absence of a ureter or the presence of more than two ureteral openings.

4. The *phenolsulphonphthalein method* (Cabot and Young[15]) is also employed in a quantitative sense. The preparation is available in ampuls. The contents of an ampul is injected into the muscle or vein; normally the urine is colored a deep red in from two and a half to four minutes. At the end of an hour the bladder is emptied and the total quantity of coloring matter estimated. This is repeated at the end of another hour and the comparison noted. Deviations from the normal are thus readily estimated. More important than this, however, is the observation of the rapidity of the elimination of the coloring matter in specimens from each kidney obtained by the ureteral catheters. Comparison of the two is indicative of the relative functional activity of the organs.

5. *Determination of electric conductivity* of the urine, introduced by Lowenbrandt (Engelmann[16]), is employed when only small quantities of urine are available. A description of the apparatus cannot be submitted here (see No. 16 of bibliography).

6. *Experimental polyuria* consists in the administration of large quantities of fluid, and measuring the quantity of urea contained in the urine at stated intervals. The urine is obtained from each kidney by ureteral catheterization. The catheters must be left *in situ* for from five to six hours; this is objectionable.

7 and 8. *Cryoscopy* of the urine in conjunction with that of the blood is of considerable value (Kümmel[17]).

To estimate the molecular concentration of a fluid, its freezing point or cryoscopic index is determined. When renal insufficiency exists, the accumulated waste products in the blood increase its molecular concentration with a corresponding fall of the freezing point. The index of the blood is .56 deg. below that of distilled water. If it

falls to .03 deg. to .05 deg., it shows renal insufficiency, and, if much lower, nephrectomy is contra-indicated. The freezing point of urine is from 1 deg. to 2.3 deg. below that of distilled water. This wide variation depends upon its organic and saline composition. However, if, under an ordinary régime of fluids and solids, the index falls more than 1 deg. below that of water, renal insufficiency may be assumed. This is much less accurate than is blood cryoscopy.

Congenital Malformation of the Kidneys and Ureters.— Congenital malformation of the kidneys relates to their *form,* their *position,* and their *number.*

Variation in the *form* of the kidney is not important. It usually consists in persistence of the *fetal kidney,* which retains its lobular conformation. It is probable that, as life goes on, kidneys of this sort are more likely to be the seat of pathological processes than obtains in the normal; this applies especially to tuberculosis (Kümmel[1]).

Double kidney, i. e., coalescence of the two organs, is the most frequent anomaly encountered; usually, at least two ureters are present.

In *horseshoe kidney* the lower poles of the organs usually merge into one another. The point of contact is in front of the vertebral column, so that the general conformation corresponds to a half moon, with its concavity upward; the reverse is very rare. The pelves are located on the anterior surfaces of the organs and their blood vessels are anomalous. Morris[18] presents a statistical study of the subject.

Unilateral kidney is rare. In this event both kidneys lie superimposed on one side, i. e., the lower pole of the upper merges with the upper pole of the lower. The ureters do not usually cross but arise from opposite sides of the kidney.

In *shield kidney* the organs are widely merged in an almost circular form, so that the total organ almost appears as one. Usually two ureters arise from the anterior surface of the malformed kidney tissue.

Hypoplasia of the kidney is of importance on the ground that one organ is likely to be *rudimentary* or *absent* (aplasia).

The *rudimentary kidney* may be the size of a walnut or smaller. The functionating parenchyma may be normal in construction or may be replaced by fibrous tissue. The ureter may be very small, or obliterated, or absent. Only the lower segment of the ureter may be present; this suggests obvious possibilities in connection with ureteral catheterization.

In *congenital absence of a kidney,* the one present is usually normal in form. Of 3370 persons sectioned, two per cent had only one kidney

(Morris[18]). As a rule the other kidney becomes hypertrophied as the individual develops.

Hyperplasia of the kidney is less frequent than the reverse. Occasionally one or two adventitious kidneys are formed.

Congenital malposition of the kidney is not uncommon. Its firmly fixed position differentiates it from movable kidney. Pathological processes involving kidneys of this sort are responsible for confusing clinical pictures, which are often not cleared up until the organ is exposed. Surgical attack is also made difficult by the fact that malplaced kidneys of this nature are likely to be adherent to the aorta or vena cava.

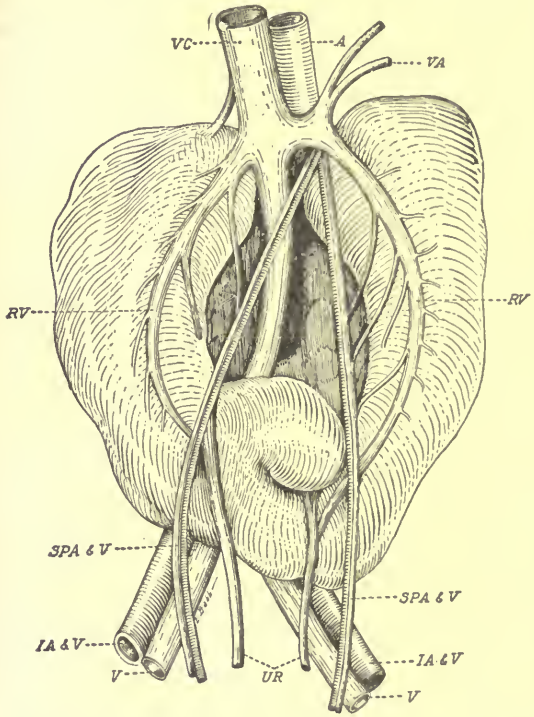

FIG. 988.— TWO KIDNEYS BLENDED, AND OCCUPYING A MEDIAN POSITION.

A, Aorta; *V C*, vena cava; *V A*, vena adiposa; *R V*, renal vein; *S P A* and *V*, spermatic artery and vein; *I A* and *V*, iliac artery and vein; *UR*, ureters; *V*, iliac vein (after Haller).

Surgical therapy in horseshoe kidney is limited to excision of the diseased sector of kidney tissue.

Congenital malformations of the ureters are divided into two forms:

1. Double ureters with normal openings do not possess surgical importance.

2. Double ureters with adventitious openings of the supernumerary ureters are of importance. The latter (in the male) are classified by Kümmel[1] as opening (a) into the bladder at an abnormal situation outside or within the sphincter area; (b) into the urethra, where the opening is always in the prostatic urethra; (c) into the seminal passages — vesicles, ejaculatory ducts or vas deferens.

Varied as the anatomical conditions are, the clinical consequences are always the same, i. e., difficulty in the discharge of urine and dilatation of the ureters and pelves, with pressure atrophy of the kidney parenchyma (uronephrosis).

In the *female,* the adventitious ureters open into the bladder, urethra, vagina, vestibule, or a persistent Gartner's tube.

Considerable clinical importance is attached to supernumerary ureters which dilate between the muscularis and the mucosa of the bladder and are responsible for tumorous protrusions into the cavity of the bladder, being likely ultimately to cause pressure on the normal ureteral openings. Still more manifest disturbances, consisting in interference with micturition and retention of urine, develop when the dilatation occurs in an adventitious ureter which opens into the urethra.

The *diagnosis* is possible only when patients afflicted with inexplicable disturbances of the urinary apparatus are subjected to an examination which includes cystoscopy and ureterocystoscopy, the introduction of shadow producing ureteral bougies, pyelography, the injection of collargol and subsequent Röntgenography.

The *treatment* in women consists in the following measures:

1. Epicystotomy. Introduction of a sound into the ureteral opening in the urethra. Division of the vesical wall on the sound. Extirpation of a transverse sector of the peripheral stump and cauterization.

2. Separation of the vaginal stump of the ureter and its implantation into the bladder.

3. Extraperitoneal implantation of the ureter into the bladder through an anterior colpotomy (see utero-urethral and vaginal fistulae, p. 2236).

In the male, suprapubic transplantation of the ureter into the bladder is the method of choice.

Injuries of the Kidneys and Ureters.— The mechanism of *subcutaneous injury of the kidney* is not always easy to understand. Solution of continuity of the organ is caused, not only by violence directed against its immediate vicinity, but also by force applied to distant parts of the body. It would appear to be certain that the kidney may be injured both by *direct* and *indirect* violence. Tuffier[19] explains the latter on the basis of commotion, or *contre coup,* which occurs in the manner described in connection with the brain and spinal cord. It is easy to see that the anatomical construction of the kidney favors the occurrence of this sort of injury. To this conception may be added

that of Küster,[20] who accounts for indirect injury to the kidney on the basis of the *bursting effect of hydraulic pressure,* a causative factor seemingly proved by experimentation.

The extent and degree of kidney injuries varies widely; they may be classified as follows:

1. *Injuries of the perirenal fat and the capsula propria* without involvement of the kidney parenchyma. These are usually limited to the formation of hematomata, which are gradually absorbed or undergo cystic degeneration.

Fig. 989.— Traumatic Rupture of Lower Pole of Kidney.

The injury was produced by a fall on the abdomen; showing the bursting effect of hydraulic pressure (F. J. Echeverria).

2. *Focal contusions of the substance of the kidney* with effusion of blood, without visible solution of continuity of parenchyma; or those associated with transverse, linear or stellate lacerations not extending into the pelvis. These usually do not provoke disturbances, but occasionally become infected and result in abscess formation.

3. *Deep lacerations extending into the pelvis and involving the kidney parenchyma in various directions* (transverse, oblique, radial, etc.) *and to a varying extent;* the latter may consist in a moderate tear or complete separation of the kidney into two or more parts. This class of injury is always attended with more or less severe bleeding and bloody infiltration of the surrounding tissues, i. e., into the perirenal and retroperitoneal fat, along the colon, into the mesentery of the small gut, along the spermatic vessels into the scrotum, the vulva, the small pelvis, and into the wall of the abdomen. The hemorrhage of itself may involve danger of a fatal outcome and cause serious disturbances, the result of pressure on the ureter and bloody distention

of the bladder. In some instances, the injury to the ureters is responsible for the accumulation of large quantities of urine within the fatty capsule, thus forming a fluctuating tumor, which often develops putrefactive and purulent infection and suppuration.

4. *Complete crushing of the kidney* (rupture), in which the organ is torn into numerous pieces more or less separated from one another. In these cases death from primary hemorrhage is not uncommon.

5. *Severe injury to the perirenal parts* with complete avulsion of the blood vessels, the pelvis or the ureters. This is very likely to be promptly followed by a fatal outcome.

6. *Traumatic nephritis.* Whether injury to the kidney is responsible for nephritis in the healthy individual is not certain. Senator[21] questions a causative relationship in this connection.

The primary *clinical picture* of kidney injuries is usually dominated by collapse or severe shock. On the other hand, it is also true that direct and indirect injuries of the kidney, with considerable solution of continuity of tissue, occurs without marked general disturbances. It is of course difficult to say whether, in a given case, shock is due to involvement of the kidney or to injury of other parts, especially as the provocative force with respect to the kidney must of necessity be severe.

Pain is an inconstant symptom, but is usually severe. Increasing pain is due to progressive distention of the lacerated tissues by blood, or urine, or both. The pain is likely to radiate into the testicle and the thigh. Whether increasing pain indicates intra- or extracapsular distention is not certain; this is unfortunate.

Visual signs of kidney injury, such as contusions of the skin, bloody effusion, and tumor may be present; in this event they are of great diagnostic significance. Rapidly increasing swelling indicates accumulation of effused blood and urine in the perirenal fat and the pelvis of the kidney (hemonephrosis, uronephrosis, and traumatic pseudonephrosis). The appearance of bloody infiltrate in the inguinal region, scrotum, etc., does not occur until several days after the injury.

Hematuria is the most important symptom of injury to the kidney. Unfortunately it is absent in cases of slight bleeding, and when the blood coagulates in the pelvis or ureters; often it does not appear until hours after the injury. The expulsion of long clots formed in the ureter is attended with severe colicky pain, finally; the passage of clots through the urethra is also very painful.

When bleeding is active the blood flows rapidly into the bladder and provokes the desire to urinate; often pure blood is passed.

In these instances, removal of blood from the bladder by catheterization and lavage is a tiresome but very necessary duty. In other instances, the quantity of blood in the urine is so slight that it can be recognized only with the aid of the microscope or by chemical examination.

Urine containing blood from the kidney is uniformly discolored; in vesical bleeding the last passed is usually the most deeply stained. Lavage of the bladder is attended with the temporary return of clear fluid when blood emanates from the kidney or ureter. This is, of course, uncertain; the cystoscope may clear up the doubt, provided the bladder can be kept clean long enough to enable the surgeon to see.

Hematuria is always attended with albuminuria; however, a nephritic kidney may be traumatized. The question of traumatic nephritis must be taken into account. Despite Senator's viewpoint, Menge[22] has shown that ungentle palpation of the kidney may provoke albuminuria, i. e., *traumatic albuminuria* may occur without hematuria.

As the kidney is at times completely macerated, the immediate picture may be one of profound collapse which is more or less recovered from, to be followed by the progressive development of acute anemia. If this happens without commensurate expulsion of blood per urethra, the inference is that the kidney is torn from its pelvis and one must search for an increasing tumor in the flank.

Secondary hemorrhage following infection of an injured kidney is not infrequent and originates from the dissolved putrefactive thrombi; this occurs often enough in apparently convalescing patients, and converts a hopeful picture into an exceedingly menacing one.

Anuria and oliguria are uncommon early complications of kidney injuries. The effect of reflex influences in this connection has been definitely demonstrated. Reflex anuria, the outcome of injury to one kidney, has been studied in connection with operative attack upon a single organ (Morris[23]). Anuria is, of course, responsible for fatal uremia.

Aside from hematuria and uremia, *extravasation of urine* is responsible for grave disturbances which carry out an accord with the amount of urinary infiltrate. Spontaneous absorption of extravasated urine occurs only when this is small in quantity and normal in character. When the perirenal tissue is invaded, the infiltrate may be circum-

scribed; when, however, the surrounding tissues are infiltrated, the extravasate, at times, involves enormous areas of the body. As a general rule, injury to the kidney parenchyma is attended with only slight infiltration of urine, while solution of continuity of the pelvis or ureter is followed by extensive extravasation. DeQuervain,[24] who presents an exhaustive communication in this connection, considers that the greatest menace in this class of cases lies in simultaneous laceration of the peritoneum, and the entrance of urine into the peritoneal cavity.

Incomplete healing of traumatic kidney lesions is attended with the persistence of urinary fistulae. In a certain number of instances, the injury causes malposition of the kidney, which results in kinking or stricture of the ureter, and these become responsible for *primary and secondary traumatic uronephrosis.* Circumscribed areas of bleeding may form the nuclei of kidney stones, and, occasionally, an area of this sort becomes the site of subsequent malignant disease. On the whole, one may say that injuries of the kidney are likely to be responsible for a number of serious conditions.

In the *diagnosis,* the clinical picture as related must be subjected to careful study. In difficult cases doubt may be cleared up by cystoscopic and ureterocystoscopic examination; these should not be omitted when the condition of the patient warrants their employment. Failing in this, operative exposure of the lesion is justified, especially when involvement of the peritoneum is suspected.

The *treatment* in injuries of the kidney, in severe cases, is directed toward relief of shock and the arrest of bleeding. In combating shock the surgeon must bear in mind that the lowered blood pressure consequent to the hemorrhage has some hemostatic value and that this must not be overcome by overstimulation, involving as it does the danger of increased loss of blood. The reader is reminded that shock, in certain instances, may be regarded as conserving the total quantity of blood.

Of course, the real surgical indication consists in exposing the seat of hemorrhage, and its prompt arrest. As a rule, this is accomplished only by nephrectomy. However, in a certain number of cases the desired end may be attained by tamponade of the wound in the kidney. This is also occasionally permissible in cases of extravasation of urine. However, when this is done, secondary nephrectomy becomes necessary in a not inconsiderable number of cases in which the immediate threatening conditions are met by tamponade (Keen[25]). In most instances the kidney is approached by the lumbar route, although, if there is

evidence of an intraperitoneal effusion, transperitoneal approach may be made (Küster[20]).

In *lumbar exposure of the kidney* technical difficulties do not arise until the fatty capsule is opened and the hemorrhagic area is reached. It is now necessary to decide quickly upon the subsequent steps and to execute them with promptness. If the bleeding is so severe as to require ligature of the renal vessels, conservatism is not longer possible, and nephrectomy is imperative. When small portions of the kidney, at either pole, especially the lower, are avulsed, a repair by suture is justified. Lacerations of the kidney may also be repaired by suture. Incomplete separation of the pelvis may be repaired in a similar manner. Unfortunately a not inconsiderable number of kidneys repaired in this way ultimately must be removed. Of course, drainage is essential in all cases.

Open Injuries of the Kidneys.— These are much less frequent than the subcutaneous. They are usually stab, incised or gunshot wounds.

Stab and incised wounds of the kidney are produced by suicides (Hari kari) or with the bayonet, sword, lance, and, occasionally, with a nail, scissors or scythe.

Suspicion of kidney involvement is aroused by the direction and character of the wound, and this becomes a certainty when blood appears in the urine or when urine flows from the opening.

In large wounds the kidney is very apt to be prolapsed through the opening. There is a predisposition to this because of the loose attachments of the organ, which is readily forced through the wound by the influence of the spasmodic contraction of the diaphragm and abdominal muscles. At times, the kidney is herniated through the wound at the time of its infliction, and remains outside with its hilus strangulated by the muscle and fascial fibers. Unless relief is afforded, the kidney becomes gangrenous.

Simple wounds of the kidney readily heal primarily. Infection may, of course, lead to perinephritis and nephritic suppuration.

Stab wounds of the kidney are likely to be associated with similar wounds of the pleura, lung, and diaphragm. In this event, *transpleural repair* of the entire lesion is the procedure of choice.

The *treatment* of open wounds of the kidney varies with their character. A number of them are best left undisturbed. Severe bleeding demands exposure of the kidney and arrest of the hemorrhage; in extensive injuries nephrectomy becomes necessary. The indications

in this connection are similar to those taken up with subcutaneous injuries.

Gunshot wounds of the kidney alone, as is very easily understood, are very rare; in almost all instances the lung, liver, spleen, gut, spinal cord, etc., are also involved.

Wounds produced by projectiles are very likely to be severe, i. e., *bursting wounds;* the mechanism of the injury, the *symptoms* and *diagnosis* are similar to those discussed in connection with subcutaneous indirect injuries.

In the *treatment* the question of the method of approach, i. e., transpleural, transperitoneal or lumbar will depend upon the character of the injury and the history of the direction in which the projectile entered. At best, the problem is an involved one and requires careful study of the picture presented in each instance.

Injuries of the Ureters.— The pelvic attachment of the ureter is most frequently injured; however, even this is uncommon. The tube is likely to be torn in severe crushing injuries, which also damage other parts and organs. As can be imagined, the character of an injury of this sort would be likely to be immediately fatal.

Isolated injury of the ureter is followed by gradual extravasation of urine, which forms a tumor in the vicinity of the torn tube. The nature of the contents of the swelling is made certain by aspiration. Injuries of moderate degree, with spontaneous healing, may be followed by cicatricial obstruction of the ureter and the subsequent development of uronephrosis.

In the *treatment* the following procedures are available: (1) Early incision of the ureter, which is indicated by local swelling, lessening in the quantity of urine, and retention of urine in the renal pelvis. (2) A policy of expectancy when the symptoms are indefinite, and not menacing. (3) Retroperitoneal exposure of the ureter when extravasation of urine and bloody infiltration or suppuration is manifest. (4) Nephrectomy in irreparable injury (complete tear) of the ureter, in reflex anuria, and eventually, when fistulous openings persist.

Open injuries of the ureter (stab wounds) are uncommon (Tuffier[19] collected three). Occasionally a ureter is torn during a difficult labor or is caused to ulcerate by a pessary.

However, the extensive operative measures of modern times have resulted in not infrequent injury to the ureters, especially in connection with vaginal operations and perineal extirpation of the rectum.

The ureter is most liable to injury where it crosses the uterine artery opposite the internal os uteri.

The treatment is taken up in connection with operations on the ureters.

Diseases of the Kidneys and Ureters.— *Movable kidney*, as already stated, is favored by the insecure anatomical fixation of the organ. On the other hand, congenital dystopia has nothing to do with movable kidney; malplaced organs are more firmly fixed than those normally situated.

In the *etiology* of movable kidney, the fact that 85 per cent of the cases occur in women, and that the condition is as frequent in the nullipara as in the multipara, suggests that the conformation of the female torso, its function and the method of living of the female sex are important factors in this connection. The fact that the right kidney shows a greater nomadic tendency than the left may be ascribed to the lower position of the former and the fact the right side of the body is more active than the left.

Kümmel[1] quotes the work of Wolkoff and Delizen, who showed that in the male the kidneys are embedded in much deeper niches than in the female, and that in both sexes the right niche is shallower than the left.

That movable kidney most frequently occurs in connection with a certain general bodily conformation has been shown by numerous observers, who are not infrequently able to diagnosticate the presence of a movable kidney by simple inspection of the patient's body, and noting its characteristic outline. An index in this connection is established by Becher and Lennhoff (quoted by Kümmel[1]) as follows: The distance from the jugulum to the symphysis is divided by the circumference of the smallest portion of the abdomen, multiplied by one hundred. When the average figure (75) thus arrived at is exceeded, palpable kidneys are usually found. In other words the kidneys are almost invariably palpable in a slender, long torso, and are not palpable in the opposite type. The influence of lacing may be stated as follows: Firm lacing on the level of the ninth and tenth ribs lessens the depth of the kidney niche, and favors movable kidney; firm lacing on the level of the twelfth rib and lower produces an opposite condition of affairs. As a matter of fact, the women of races where lacing is not practiced are much more frequently afflicted with movable kidney than are those where this refinement of the toilet is habitual.

Freely movable kidneys are frequently observed as a part of general *enteroptosis,* in which all of the connections of the abdominal viscera are relaxed.

Trauma plays on exceedingly important rôle in the production of movable kidney. The causative factors are similar to those discussed in connection with subcutaneous injuries. That the anatomical peculiarities spoken of are strong predisposing factors in this connection is shown by the fact that 94 per cent of this class of movable kidney (traumatic) occurs in women (Küster[20]).

Recognition of movable kidney is based on *palpation* of the organ; this is accomplished in the manner stated above, where the conditions from which it must be differentiated are also mentioned.

The *clinical evidences* of movable kidney are exceedingly variable. At times, a freely movable kidney may not give rise to the least disturbance; on the other hand, moderate mobility is often responsible for distressing symptoms, the outcome of pressure on contiguous organs, especially those situated in the female pelvis. Of the symptoms ascribed to pressure, the more common are nausea, vomiting, icterus, constipation, cardiac irregularities, convulsions, singultus, esophageal spasm, and numerous other reflex disturbances. Of these, those relating to pressure on the organs in the female pelvis, including the appendix, are the most common (Edebohls[26]). It is not by any means easy to be certain whether a given movable kidney is responsible for the formidable array of reflex disturbances mentioned, or whether it is simply a part of a general enteroptosis, which is the real disturbing influence. A conclusion in this connection must rest on a careful analysis of all the factors operative in each case.

Persons afflicted with movable kidneys are likely to be subject to peculiar attacks which Dietl first described as *strangulation of the kidney* (*Dietl's crisis*). The attacks are characterized by the sudden appearance of severe pain, which gradually increases and is attended with nausea, vomiting, small pulse, cold perspiration, and, occasionally, chill and subsequent fever. Examination reveals marked tenderness in the region of the kidney; this may be so painful that examination without narcosis is impracticable. When palpation is feasible the kidney is readily felt as a round tumor. As a rule, the attack subsides in a short time (hours). Recession of the attack is followed by the sudden expulsion of a large quantity of urine.

No doubt the condition is the result of interference with the flow of urine from the pelvis of the kidney, i. e., an acute hydronephrosis is

established. However, two conditions must be taken into account, one in which the retention is the outcome of anatomical changes in the ureter or pelvis alone, and that in which the vessels also are affected. In the cases in which the movable kidney is the responsible factor, not only is the ureter obstructed, but the entire hilus is kinked and rotated on its axis, so that both the ureter and the blood vessels are affected. Thus, to the retention of urine, is added obstruction of the venous circulation, which in turn adds enormously to the acute swelling of the parenchyma of the kidney and distention of its capsula propria. In the second instance the subjective symptoms are, of course, very severe, and release of the obstruction is not followed by prompt restitution to the normal.

The *treatment of acute strangulation of the kidney* consists in bimanual reposition of the displaced kidney; this is often difficult. Failing in this the kidney must be exposed, replaced in its normal position and fastened there (*nephropexy*). If the strangulation has caused gangrene, *nephrectomy* becomes necessary.

Abdominal supports are effective only when the mobile kidney is a part of a more or less general enteroptosis. When the abdominal wall is firm and the patient is thin, neither an abdominal supporter nor an especially constructed truss and pad is of the slightest value. Supporters must be applied with the patient in the elevated hip position. Pressure on a displaced kidney is objectionable for obvious reasons; the aim of the apparatus is to maintain the reduced kidney in its normal position. In a certain number of cases retention of the kidney in its normal position, and increase of fatty perirenal tissue, results in permanent retention of the organ in its original position.

Operative fixation of the kidney (*nephropexy*) was first practiced by Hahn,[27] but with little success. Since then a number of methods, too numerous to include here, have been employed for the purpose. None of these proved of value until the principle of partial decortication and suture of the kidney, evolved from the first step in this direction taken by Lloyd,[28] was perfected. The method most widely practiced today (based on the work of Elebohls[26]) is carried out as follows: The kidney is exposed through a vertical incision at the outer edge of the quadratus lumborum muscle, which separates the fibers of the latissimus dorsi and splits the transversalis fascia; the kidney, together with its fatty capsule, is delivered, and the latter is resected in its entirety. The fibrous capsule is divided along the convex border of the kidney and separated from it for one-half of its extent on either

side; two mattress sutures (chromic gut) are placed in the flaps of the capsule on their visceral sides, so that, when the sutures are passed through the abdominal wall and tied, the everted capsule is brought in contact with the anterior surface of the transversalis fascia. It is expected that the kidney will become adherent to the site of its fixation by scar tissue. The superficial parts are sutured without drainage.

Not a few surgeons attach importance to fixation of the kidney in as near to the normal position as is possible. A number of methods have been devised intended to accomplish this. Riedel[29] exposes the kidney through an oblique incision close to and parallel to the twelfth rib, resects the fatty capsule, and opens the proper capsule transversely at the posterior surface of the organ. After placing the latter as high up as is possible, the lower portion of the capsule is sutured to the anterior surface of the quadratus lumborum. On the lateral sides the subserous fat and peritoneum are sutured to the reflected capsule. The posterior aspect of the denuded kidney and the space beneath the lower pole of the organ are packed with iodoform gauze. Finally, the edge of the sacrolumbalis and the abdominal wall are sutured over the tampons. The latter are removed at the end of four weeks.

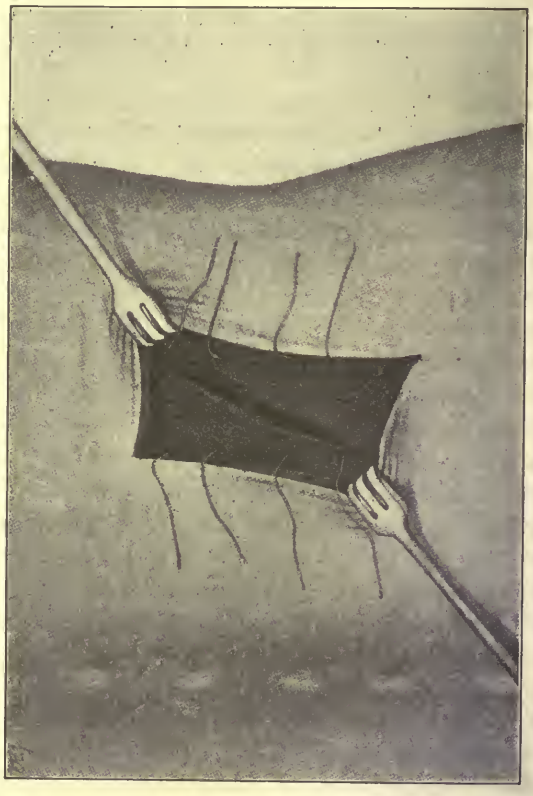

FIG. 990.— NEPHROPEXY.
Sutures in abdominal wall (Edebohls).

A number of surgeons employ the oblique incision and, after resect-

ing the fatty capsule, turn up a transverse flap of the proper capsule and suture it to the twelfth rib. Detailed descriptions of the various methods are presented by Morris[23] and Bryant.[30]

The large number of methods of fixation of the kidney justifies the conclusion that none accomplishes the purpose in all instances. The method of Edebohls fixes the kidney in an abnormal position, but it relieves pressure symptoms. In cases in which the movable kidney is responsible for kinking of the ureter, the method of Riedel[29] or one of its modifications is more likely to afford relief. The mortality of nephropexy is estimated at 1.18 per cent (Edebohls[26]). Recurrences are observed in twenty per cent of the cases (Kümmel[1]).

Retention Tumors of the Kidneys.— When the flow of kidney excretion is interfered with, the pelvis and the calices are dilated and, as the result of the increasing pressure, finally develop into cystic tumors, at times of enormous dimensions. If aseptic urine is thus accumulated the condition is designated as *hydronephrosis,* if the contents is purulent the term *pyonephrosis* is employed. Küster[20] suggests that the term *sacculated kidney* be used to designate the condition and that this be qualified in accord with its contents. Kümmel[1] pleads for the use of the terms *uronephrosis, uropyonephrosis and pyonephrosis,* which are self explanatory. Dilatation of the pelvis of the kidney may be designated as *pyelo-ectasia,* and the higher degrees as sacculated kidney or *hydronephrosis.*

Space is conserved, if at this time the consequences arising from hindrance to the egress of kidney excretion are considered. In all forms of urinary stasis this hindrance may be complete or incomplete, which leads to *open or closed uronephrosis.*

In *simple uronephrosis* the effect of back pressure of urinary retention upon the kidney and upon its function varies in accord with whether the retention is complete and permanent, incomplete or transient, or is slowly converted from an incomplete to a complete one. Guyon[31] has shown that when the flow of urine is suddenly completely interrupted, an enormous increase of fluid pressure in the pelvis of the kidney and the free portion of the ureter occurs at once. This distends the elastic pelvis and is evinced clinically by the severe pain and reflex disturbances discussed in connection with strangulaton of the kidney (see Movable Kidney). The reaction in the kidney itself consists in marked congestion (at times, hemorrhagic) and edematous saturation of the parenchyma. These circulatory disturbances are followed (after the first hour) by lessening of the quantity of the

separated urine and also by reduction in its soluble content (urea, etc.). As a rule, this reduced functional activity goes on for a long time and may be followed by sacculation of the kidney.

The latter is, however, not always the case and, occasionally, sudden occlusion of the ureter may be attended with complete arrest of the excretory function of the kidney. In this event, the microscopical changes in the kidney are not unlike those occurring in cases of acute nephritis (*pyelonéphrite aseptique* of Albarran[32]).

After the outflow is reëstablished, the residual cystic dilatation of the kidney pelvis, consequent to complete obstruction, is never as great as is that of so-called hydronephrosis. In the first instance the still persisting kidney secretion is absorbed through the cyst wall; this maintains a certain degree of balance. When the outflow of urine is reëstablished in a few hours or days a transient polyuria is established. This shows that the increased excretion of urine following strangulation of the kidney or intermittent hydronephrosis is not due to the sudden egress of retained urine.

When total retention has persisted for a long time, the reëstablished flow of urine exceeds that of the normal kidney. However, the quantity of urea thrown off is reduced to below the normal. This poverty of urea in the cystic urine is due to alterations in the secreting parenchyma. In standardizing the capacity of the obstructed kidney it must be remembered that its full function (always reduced) is not reached until after the second day following reëstablishment of urinary output.

In *uropyonephrosis* the conditions are about the same as in uronephrosis. In real *pyonephrosis* little, if any, urine is excreted. The contents may show traces of urea. If the outflow is reëstablished, the residual tissues may functionate to a slight extent.

It is conceivable that incomplete obstruction to the urinary flow has a less deleterious effect upon the kidney structure than the complete, but it is also comprehensible that the determining factor in this connection is the length of time that the obstruction persists. In complete obstruction the secretory activity of the kidney is soon arrested and no great dilatation of the pelvis occurs; in the incomplete variety the reverse obtains, i. e., the kidney parenchyma continues to functionate and the prolonged increase of intrarenal pressure progressively dilates sometimes to an enormous extent, the structures central to the point of obstruction. It is worthy of note that even greatly dilated tissue may be capable of function when an opportunity for the free

discharge of its excretion is provided (correction of the obstruction). However, this must be standardized, not by the bulk of urine it excretes, but by the total quantity of urea, etc., its filtrate contains; hence the rationale of the method of determining kidney function stated above (p. 2271). When a prolonged partial obstruction has seriously damaged the kidney tissue, a sudden complete occlusion is not attended with stormy clinical manifestations, for the obvious reason that only a slight increase of pressure is still possible.

Uronephrosis (*hydronephrosis*).—Hindrances to urinary flow responsible for uronephrosis may be located in the upper or lower urinary passages. They may be congenital or acquired; the former are taken up in connection with malformations of the ureters.

Acquired uronephrosis is due to a number of causes and, in a general way, the condition may be regarded as secondary to any determining obstruction in the urinary tract. It is manifest that peripheral hindrances, such as phimosis, urethral strictures, prostatic hypertrophy, etc., are not important factors. Aside from the occasional occlusion of the vesical portion of the ureter (scars, tumors, etc.), the most important causes of uronephrosis lie in the domain of the ureter or the pelvis of the kidney itself. Inflammatory processes in the region of the female genitals (parametritis) and occlusion by stasis are also important factors in this connection. However, in by far the greater number of instances the obstruction is due to narrowing of the lumen of the ureters, coincident to pathological processes; gonorrheal, tuberculous, and chronic proliferating ureteritis; phlegmonous ureteritis and peri-ureteritis, and papilloma of the ureter. Of these the gonorrheal is the most important. Tuffier[33] has shown that stricture of the ureter is responsible for forty-five per cent of the urinary fistulae sequential to nephrotomy. The exact pathological anatomy of this class of cases is taken up *in extenso* by Braun.[34] In a certain number of cases, uronephrosis is attributable to *torsion of the ureter*. The causative influence of trauma is established. In most instances the ureter is crushed and the subsequent scar tissue formation ultimately strictures it (Wagner[35]). In addition to this the formation of clots in the pelvis and ureter, occurring in connection with contuson of the kidney, may be responsible for uronephrosis. The causative relationship of movable kidney has already been taken up (p. 2283). Kümmel[1] presents an elaborate analysis of the mechanism of uronephrosis occurring in connection with kinking of the ureters, and its manual or spontaneous correction. Finally, the weight of the sacculated kidney

becomes so great that correction is no longer possible, and an incomplete or open (intermittent or remittent) uronephrosis becomes a complete one.

In uronephrosis due to kinking and to strictural occlusion of the ureter (in the non-dislocated kidney), the relationship of the ureter to the pelvis of the kidney is altered. The pelvis gradually dilates outward, forward, and downward, and the kidney is rotated outward on its longitudinal axis; this causes the opening into the ureter to rise upward and away from the lower pole of the kidney. That is to say, the sacculation of the kidney occurs mostly in a downward and forward direction and the ureter lies in apposition with its posterior aspect.

FIG. 991.—URONEPHROSIS FROM ITS POSTERIOR ASPECT.

The ureter enters the sacculation at its upper part.

Observation of the ureteral opening from the pelvic side of the sacculation often reveals the presence of a valvular arrangement in this situation. This is due to the angular relationship that the dilated pelvis bears to the direction of the ureter, and may occur in all forms of hindrance to the outflow of the kidney excretion.

The *causes of uronephrosis* may be summarized as *pressure, stricture, stoppage,* and *kinking.* It is probable that *swelling of the mucosa of the pelvis,* in conjunction with inflammatory processes, may be responsible for the condition. At this point it is permissible to emphasize the statement that intermittent uronephrosis does not occur alone in connection with dislocated kidney, but is also the outcome of a sudden increased demand made upon the excretory function of this organ, and, too, may be caused by swelling of the mucosa of the renal pelvis.

The *symptoms and course of uronephrosis* are exceedingly variable. The stormy clinical picture of acute strangulation of a kinked hilus or

torsion of the ureter represents the extreme in this regard; while, on the other hand, the process may, as the outcome of gradually increasing obstruction, develop into massive sacculation of the kidney with little or no disturbance, and escape notice until attention is attracted by the presence of a tumor or by pressure symptoms.

The *tumor of uronephrosis,* if the kidney is not primarily displaced, occupies the area between the lumbar region and the anterior abdominal wall and protrudes below the free borders of the ribs. Ballottement may be elicited by percussion pressure with one hand over the space of Petit and the other upon the anterior abdomen. The *consistence of the tumor* depends upon its size; large sacculated kidneys fluctuate; the smaller are likely to be tense and hard. Occasionally, the lobulated outline of the dilated calices is palpable through the abdominal wall.

As the unaffected kidney carries the burden of the urinary excretion, only the examination of a specimen of urine obtained from the pathological kidney is of diagnostic value.

In the *diagnosis* the symptoms stated and the urinary analytical findings are likely to be determining. In complete occlusion of the ureter simple cystoscopy, in conjunction with the color tests already discussed, may be all that it necessary. This is of especial importance, as catheterization of the ureter, for obvious reasons, may not succeed. If access to the sacculated kidney is attained, the latter may be injected with collargol and the conditions visualized in the Röntgenographic negative.

In doubtful cases it is permissible to *aspirate* the tumor. The maneuver, as in all organs closely associated with the peritoneum, is not without danger; it should be employed only when the surgeon is prepared to operate immediately.

The *treatment,* in the ideal, consists in measures directed toward overcoming the obstruction. For a long time, extirpation of the sacculated kidney was universally practiced. In 1892, however, a new epoch in the treatment of hydronephrosis was begun by the work of Fenger[36] (Chicago), who relieved the valvular obstruction at the ureteropelvic opening by an operative method not unlike that of the Heineke-Mikulicz pyloroplasty (p. 1977). At about the same time, Küster[37] excised the upper occluded sector of the ureter and reimplanted it into the lower portion of the distended pelvis. Since then the conservative treatment of hydronephrosis has been actively developed. It may be arranged as follows:

1. *Catheterization of the sacculated renal pelvis from the bladder* and its lavage with silver nitrate solution. This has a limited field of usefulness.

2. *Aspiration* — which is only a palliative measure.

3. *Nephropexy,* when uronephrosis is due to movable kidney.

4. *Nephrotomy,* which aims at preservation of the still existing functionating kidney parenchyma. The sacculated pelvis is freely incised and a urinary fistula is established; in a number of cases the ureter becomes patent and the fistula heals. If the ureter is completely occluded, the fistula persists. In this event, pyonephrosis is likely to supervene; the method is not largely practiced.

5. *Correction of the valvular arrangement* of the upper end of the ureter by the method of Fenger or of Küster, already referred to.

6. *Pyeloplication* (Israel[38]), which consists of obliterating the sacculation by suture.

7. *Resection of the sac* (Geiss[39]) and reëstablishment of the normal relationship of the ureteral opening to the kidney parenchyma.

8. *Opening of the sac,* location of the stricture (by bougie) and correction of the latter by excision and end to end anastomosis or by incision and ureteroplasty.

Despite this formidable series of conservative measures of relief, there still remain a number of cases in which it will be found necessary to *extirpate* the sacculated kidney.

As to the method of approach, the lumbar is used in all instances except when a dislocated uronephrotic kidney makes the transperitoneal route necessary.

Suppurative Nephritis and Suppurative Retention Tumors of the Kidney.— Suppuration of the kidney appears in a variety of forms. The process may consist in a simple purulent inflammation of the pelvis (*pyelitis*); a single or multiple abscess of the kidney parenchyma (*nephritis suppurativa*); or retention of urine and pus is added to the picture and leads to distention of the renal pelvis (*pyonephrosis*), in which pressure and secondary suppuration destroys the entire renal parenchyma, so that only a connective tissue sac remains; or a simple uronephrosis is converted into a pyonephrosis. In other instances, the presence of calculi in the kidney or its pelvis is the primary lesion, and to this is added suppuration and alkaline fermentation; or again, the inflammatory process extends to the kidney from its surroundings, or it may be tuberculous. When to this is added the fact that these several processes may exist alone, or in

combination, or merge into one another, it is comprehensible that suppuration of the kidney is, to say the least, likely to present a kaleidoscopic clinical picture.

Renal suppuration develops in three ways; directly through the blood (*descending hematogenous infection*); from the bladder (*ascending urogenous infection*); or by way of the lymph channels from contiguous organs (*lymphogenous infection*). Of the three, it is probable that the first (hematogenous) is the most frequent.

1. *Hematogenous descending infection* is easily understood, when it is remembered that the kidney is one of the chief organs concerned in elimination of pathogenic bacteria. However, it is probable that the kidney is not damaged in this connection, unless some intercurrent affection, such as urinary stasis, etc., creates conditions favorable to the multiplication of bacterial life. Of the infectious diseases, the following are most frequently responsible for the infection: Measles, scarlatina, smallpox, typhus, tuberculosis. Of the bacterial excitants, bacterium coli, staphylococcus and streptococcus are the most common invaders. The renal pelvis is especially susceptible to cholera, dysentery, typhoid, variola, diphtheria, scarlatina, measles, and influenza.

The *pathological anatomy* of these invasions consists in large and small foci of infection lying on the cortex of the kidney, the blood vessels of the foci being striped with collections of bacteria. The individual focus is usually wedge shaped, its base lying under the capsule. The subsequent changes are similar to those occurring in connection with acute infectious inflammatory processes elsewhere in the body and ending in abscess formation. A number of small foci coalesce and form one large abscess. Finally, the entire kidney may consist of a system of confluent abscesses lying between the trabeculae of the kidney, with most of the parenchyma destroyed. The fatty capsule is contracted and firmly apposed to the surface of the kidney; or an abscess breaks into the fatty tissue and is either isolated there or extends, invading the retroperitoneal tissues, where it forms a paranephritic abscess. If pus invades the renal pelvis, it is discharged in the urine (*pyuria*). The pelvis and the ureter are infected, become swollen and interfere with the flow of urine. In other instances, the dilated kidney sectors do not communicate freely with the ureters, being cut off from the latter by the same valvular arrangement as occurs in connection with uronephrosis. The inflammatory exudate and the urinary secretion are retained and an enormous sacculation of the kidney is developed; the parenchyma is finally destroyed.

2. *Urogenous infection,* despite the fact that it must extend "against the stream," undoubtedly occurs. The mechanism of this ascending infection is explained on the basis of antiperistaltic movements on the part of the ureter when it is overdistended. In other cases, inflammatory involvement of the ureteral opening in the bladder abolishes the sphincteric control in this situation, and the infection extends along the ureter into the kidney. In the latter situation the subsequent changes are closely related to those occurring in connection with hematogenous infection.

3. *Lymphogenous infection* of the kidney is conveyed to the organ through the lymphatic vessels of the perinephritic fat. This is most often the outcome of infection carried from the colon (bacterium coli). The existence of lymphatic connection between the colon and the kidney has been shown.

The *clinical symptoms* of purulent pyelitis and pyonephritis are likely to be characteristic. Embolic infection is attended with severe pain ascribable to the concomitant hyperemia and edema of the entire organ. The attack of pain is attended with the presence of large numbers of bacteria in the urine. When the ureter is infected, the pain radiates along its course and is likely to suggest appendicitis. The temperature curve and the constitutional disturbances carry out an accord with the character of the infection; these are much increased when the urinary excretion distends the kidney capsule. Palpation reveals an enlarged, tender kidney. The examination of the urine is, of course, of great importance, furnishing, as it does, information as to the character of the infectious process.

The *diagnosis,* early in the process, is not always easy, especially when the kidney infection occurs in the course of an acute infectious disease, the symptoms of which may dominate the picture. In these cases, pain, tenderness, tumor, and the urinary findings must be relied upon.

Of course, catheterization of the ureters is of great value and determines involvement of the renal pelvis and the presence of urinary stasis. Albuminuria is only diagnostic when the urine is acid. The albumin content of the urine is dependent upon the quantity of pus cells. A leukocyte content of 50,000 to 70,000 corresponds to 1 per cent Esbach albumin.

The *treatment* varies with the condition of the kidney. When only the ureter and the renal pelvis are affected, lavage of the latter is permissible when medication (urotropin, hexal, cystopurin, helmitol) and

the drinking of large quantities of water are ineffective. The measure, followed by the injection of 20 to 50 gm. of a 1 per cent silver nitrate solution or of collargol, is particularly efficacious in acute pyelitis. In the chronic form, when the pelvis and the calices are dilated and the kidney parenchyma is in a condition of interstitial nephritis, lavage of the pelvis does not accomplish the intent. In these cases, serological treatment, though not often effective, is justified.

When the kidney itself is affected, only *operative measures of relief* are worthy of consideration. The organ is exposed (lumbar route) and delivered through the wound. Further procedures are decided in accord with the findings. It is important to determine the character of the infection before the kidney is exposed, on the ground that, when the bacterium coli is the aggressor, conservative methods of procedure are most likely to be successful. It is impossible to determine this macroscopically in the exposed kidney.

The following methods of procedure come under consideration:

Decapsulation, alone, is permissible when the capsule is very tense, the subjacent parenchyma is intact, and urinary examination shows few pus cells and a large number of bacteria. In the presence of a few small abscesses, these may be opened. To permit rapid subsequent nephrotomy (when the symptoms do not promptly subside) the wound of approach is tamponed.

Nephrotomy, in suppurative nephritis, is followed by relief in a considerable number of cases, despite the fact that it is not possible to be certain that all the foci have been opened. The method involves the danger of severe bleeding. In all instances the kidney wound should be tamponed. When inspection of the sectioned kidney reveals a restricted lesion, this may be resected and the residual cavity tamponed; occasionally, a cavity of this sort may be sutured.

Extirpation of the kidney is indicated when the parenchyma is extensively involved. In these instances, if the sister organ is healthy, procrastination is unwise. When the reverse obtains, the surgeon must content himself with nephrotomy; this has, in not a few instances, been performed on both kidneys with success (Morris,[23] Israel,[38] Lennander,[40] Kümmel,[41] and others). In the presence of pyonephrosis a search must always be made for the causative constriction, and an effort made at correction.

Renal and Ureteral Lithiasis.— When a foreign body invades the urinary passages it becomes encrusted with the salts held in solution by the urine. Compared to the total number of urinary calculi, this

may be regarded as a rare occurrence; on the other hand, it indicated the path which led to an understanding of the *etiology* of the development of urinary calculi. The history of the laborious steps taken in this direction cannot be produced here. A review in this connection

FIG. 992.— CALCULI WITH PYONEPHROSIS AND GANGRENOUS CYSTITIS (Symmers, Bellevue Hospital case).

is presented by Kümmel.[1] While it is probable that the domination of certain chemical constituents of the urine may have a causative influence in the development of calculi, the most important factors are all those conditions which favor stasis and retention of urine, i. e., the conditions which make possible the development of urinary calculi

exist in the normal urine, and delay or arrest in expulsion of the latter is all that is necessary to provoke sedimentation of its salts, and calculus formation follows. This is borne out by the curious fact that calculi are almost invariably found in the urinary passages of patients afflicted with traumatic paraplegia.

While completely formed concretions are found in all portions of the urinary passages, they are usually formed in the kidney, its pelvis and the bladder. Small brick red deposits are found in the kidney of 47 per cent of the newborn (uric acid infarcts); these soon disappear. Somewhat larger concretions are called *kidney sand;* still larger ones (size of barley corn) are designated as *kidney gravel.* Concretions which the urinary organs are capable of expelling are grouped under the general term *gravel,* the term *calculi* being reserved for larger concretions which cannot be expelled. Concretions in the renal pelvis and bladder may reach enormous dimensions.

While calculi occur in all portions of the kidney, they develop most frequently in the calices and the pelvis; their conformation usually corresponds to the situation in which they are formed. Those found in the kidney are usually the size of a pea; in the pelvis they are larger, and in the ureter their outline is likely to be ovoid or cylindrical. Stones in the pelvis may form a complete cast of this part or may be multiple and faceted. Calcareous incrustations of inflamed areas usually consist of phosphate of lime and ammonia. As can be imagined, these incrustations at times take on grotesque forms.

The *consequences of harboring kidney stones consist* primarily in irritation of the renal parenchyma, which responds in the form of chronic inflammatory and degenerative processes. The severity of these changes depends upon whether the urine remains aseptic or becomes infected and, in the latter event, undergoes alkaline fermentation and is associated with purulent pyelitis and nephritis.

In the first instance, the process is restricted to contractural changes, to which must be added the result of retention of urine. The degree of the latter is of course dependent upon the location of the stone; the extreme of this is represented by uronephrosis (p. 2287).

However, in most cases renal lithiasis is attended with *secondary infection* and the development of *suppurative pyelitis* and *nephritis.* Thus the kidney becomes the prey of infection and irritation combined with obstruction of the urinary passages, the total of which is the picture of pyonephrosis of a severe form attended with formation of large abscess cavities.

The infection may be hematogenous or may ascend from the lower urinary passages; the latter is the more frequent and is most often gonorrheal in character. Infections occurring in connection with calculi are often responsible for perinephritic abscesses.

Prolonged inflammatory processes in one kidney always affect the sister organ in some way. Even a simple aseptic sclerosis of a kidney stimulates the other organ into hypertrophic changes. However, *only a sound kidney is capable of compensatory hypertrophy.* In cases of renal lithiasis, the integrity of the opposite kidney is constantly endangered. Yet more unfortunate is the fact that the disposition to form calculi is not limited to one kidney and in many instances both organs are affected or, if only one is affected, the disposition of the other is to emulate the example of the sister kidney.

Most menacing of all are the conditions when infection has occurred; then the ureter and the bladder are soon invaded and the tireless pyogenic aggressors ascend on the opposite side to duplicate in the sister kidney the havoc already wrought in the organ primarily affected. Morris[23] presents a statistical review in this connection.

Kümmel[1] divides renal calculi into *primary,* which develop from sedimentation of the constituents ordinarily held in solution in the kidney, and *secondary,* those which develop as the outcome of pathological changes in the renal pelvis and calices, and are formed in connection with inflammatory exudates.

The symptomatology of renal lithiasis, aside from the Röntgenographic findings, does not furnish us with a single characteristic sign.

Pain varies in accord with the motility or the fixed position of the stone, the influence of the latter upon the flow of urine, its size, and whether it gains access to the ureter or not. Pain is also largely influenced by the presence or absence of infection.

In the cases following a moderate course, pain is heavy and indefinite, but is likely to be increased by muscular exertion, riding, etc. Occasionally forcible percussion in the region of the kidney is attended with severe pain.

When the stones are small, pain is likely to take on the form of *renal colic.* This phenomenon, long regarded as pathognomonic of stone, is attributed to the passage of a calculus through the ureter into the bladder. The attack is attended with very severe pain which radiates toward the bladder, the testicle, the glans, the inner side of the thigh, the heel, the abdominal wall, the stomach and the pelvis. As a rule, renal colic develops suddenly (even during sleep) and may

be so severe as to cause syncope and collapse. The patient usually vomits and is covered with sweat; the pulse is small and rapid. The attack is attended with urinary tenesmus, while only a scant amount of dark, often bloody, urine is expelled; or the bladder simply discharges thick, bloody mucus (pyelonephritis). Occasionally, in unilateral renal lithiasis and complete occlusion of the ureter, the sister organ excretes a large quantity of clear, light colored urine (urina spastica). The expulsion of gravel is attended with the presentation of a less stormy picture.

It would be permissible to regard these attacks as characteristic of the passage of a foreign body through the ureter, were it not for the fact that they occur in connection with *all conditions which increase the tension of the capsule of the kidney;* these include acute inflammatory congestion, venous stasis (strangulation) and urinary stasis in the renal pelvis, the outcome of interference with the flow of urine irrespective of the causative factor. Renal colic is, therefore, not by any means indicative of lithiasis.

Hematuria, though an important symptom, is also, at times, deceptive; it may be microscopical in quantity, or the urine may appear to consist of pure blood. In standardizing its significance one must remember that it also occurs in connection with renal tuberculosis, tumors, pyo- and uronephritis, and a certain kind of nephritis to be taken up later.

The *elimination of calculi* or their fragments, sequential or coincident to colic, is a positive sign of nephrolithiasis.

Symptoms of bladder irritation occur in nephrolithiasis as often as in cystitis, and attend the former when the bladder is normal. The use of the cystoscope has reduced the number of cases of cystitis seen by the urologist one half.

Anuria argues strongly for nephrolithiasis. This often means that one kidney has been inoperative for a varying period of time and that the second is suddenly blocked by a stone.

Reflex anuria is accepted as such by a number of careful observers. In this connection it is interesting to note the statement of Kümmel,[1] as follows: "I, personally, in my not inconsiderable material, have never seen a case of true reflex anuria." He offers an extended analysis of the various aspects of the situation. It would seem that the occurrence of reflex anuria in a healthy organ, in connection with a lesion in a sister kidney, is somewhat doubtful. On the other hand, it undoubtedly occurs as the outcome of peripheral irritation (bladder,

urethra, etc.), and is then ascribable to nervous influences. One may be justified in assuming that anuria indicates bilateral kidney lesions in most, if not all, instances.

The *diagnosis* of renal lithiasis may be entwined with many difficulties. Aside from the fact that ureteral catheterization, the Röntgenographic findings, and the clinical picture may be misleading with

FIG. 993.— RÖNTGENOGRAM OF A LARGE STONE IN THE PELVIS OF THE KIDNEY (Harlem Hospital case).

regard to the exact character of the kidney lesion; the question of differentiation with respect to affections of other organs, such as biliary colic, must be taken into account.

Pain in the kidney and bladder regions, hematuria and pyuria arouse suspicion of stone, yet, conclusion in this respect is permissible only when the results of the following are carefully analyzed: the history

of the case; palpation; examination of a specimen of urine obtained by sterile catheterization of the ureters; and the Röntgenogram.

Of these, the Röntgenographic findings, if possible, are of the greatest help. While negative findings are not determining, the recent improvements in technic usually make discovery of a kidney stone by this means a comparatively easy matter. The method is materially aided by preliminary injection of the renal pelvis with collargol, which outlines the pelvis and saturates the concretions with silver salt.

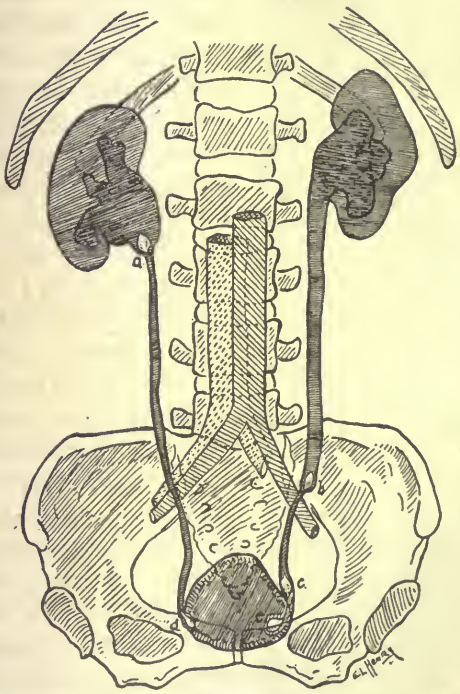

FIG. 994.—WHERE URINARY CALCULI ARE MOST LIKELY TO LOCALIZE.

a, Stone at neck of the renal pelvis; *b*, stone in the ureter where it enters the true pelvis; *c*, stone in the ureter before it enters the wall of the bladder; *d*, stone in the ureter in the bladder wall; *e*, stone elevating the bladder mucosa.

In all instances it is essential that the surgeon be assured that the patient possesses two kidneys, and as to the extent of the functional activity of the organs.

Only the *operative treatment* of nephrolithiasis is considered here. Removal of a stone from an otherwise healthy kidney was first accomplished by Morris[43] (1888). Today, the principle of removal of renal calculi, in lieu of nephrectomy, is established. It is always the method of choice when infection is absent; when the latter is present, the problem must be given careful consideration before the method of procedure is decided upon.

The question as to whether all calculi, especially when infection has not occurred, should be removed has been thoroughly studied. One is justified in stating that, while quiescent nephrolithiasis does not imperatively demand removal of the concretions, the dangers to the kidneys, involving as they do the possibility of such serious con-

sequences, render their removal advisable, unless a direct contra-indication to the operation exists.

When infection has developed, there is no doubt that operative measures of relief are fully justified, and that delay in this connection is not permissible. This is especially true in calculus anuria, in which event speedy relief must be afforded. For the technic see Operations on the Kidneys and Ureters (p. 2326).

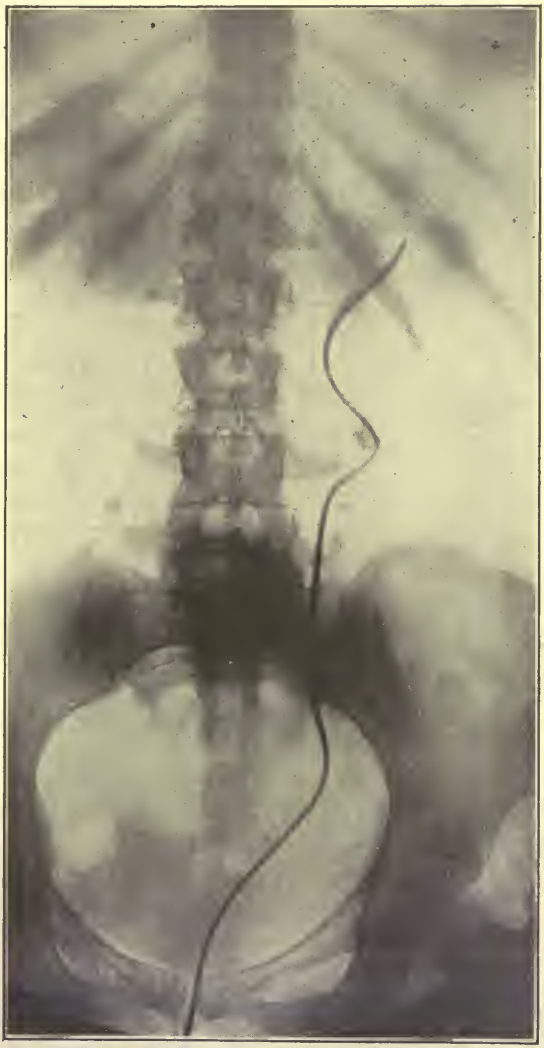

FIG. 995.—RÖNTGENOGRAM OF A STONE IN THE URETER.

The location of the stone is indicated by the shadow catheter (Beer).

Ureteral Calculi.—Calculi are rarely formed in the ureters; they are usually kidney stones arrested during their journey toward the bladder. Occasionally, a ureteral stone develops at the site of a pathological condition in the mucosa, i. e., a small concretion forms and this is added to by the deposit of urinary salts.

Ureteral calculi may be *single* or *multiple;* as the stones usually descend from the pelvis the presence of farther concretions in the latter situation must be sought for. The *form* and *size* of ureteral calculi varies widely

(from that of a pea to that of a lemon); most of them are spindle shaped. The constituents of the calculi are similar to those of the kidney. Most of them have an oxalate of lime nucleus.

The *seat of ureteral stones* is very important. Favorite locations are at the narrowed points of the lumen of the ureters, as follows: near the neck of the renal pelvis; where the ureter crosses the linea arcuata and enters the true pelvis; and where the ureter enters the bladder. In addition to this, stones often rest in the portion of the ureter traversing the bladder wall. Between these points of narrowing the ureter is spindle shaped. Aside from its normal points of narrowing, the ureter may be strictured elsewhere, as the result of ascending infection; or kinked; or it may be compressed by contiguous tumors, or by extrinsic inflammatory processes; all of which favor the formation of concretions.

The *symptoms* of ureteral calculi do not vary widely from those presented in connection with nephrolithiasis. None of these are distinctly indicative of the location of the stone in the ureter. The passage of a small oval stone per urethram indicates the cause of a preceding colic. Bleeding is slight and may not be discovered, except with the microscope.

The *diagnosis* of ureteral calculus is the most difficult problem in the study of lithiasis of the urinary passages.

Ureteral catheterization is helpful, but is not a certain diagnostic measure. Arrest of the catheter may be due to other causes (stricture); or the stone may permit the passage of the instrument by its side; or the stone may be propelled into the renal pelvis.

Radiography is of diagnostic value with the same restrictions mentioned in connection with nephrolithiasis; it is the most certain sign we have. In doubtful cases the introduction into the ureter of a "shadow sound," and subsequent radiography, may decide the question (Fig. 995). If the radiogram is preceded by a collargol injection, the conformation of the ureter and renal pelvis may also be determined.

The *consequences of ureteral lithiasis* are not unlike those occurring in connection with stone in the kidney. The protracted presence of a stone in the ureter leads to urinary stasis which, in turn, is responsible for dilatation of the passages central to the obstruction. As is the case in the kidney, this is ultimately supplemented by infection. The ever lurking bacterial hyenas (coli, staphylococci, etc.) soon find their way (by the blood) to the site of the damaged urinary pas-

sage and by their usual method of destruction become responsible for pyelitis, infective pyo- and uronephrosis, and abscess formation.

The *greatest danger* to the patient occurs in cases of *bilateral ureteral calculus occlusion* with consequent *anuria*. The consequences of this misfortune are discussed in connection with renal urinary stasis. This sounds a warning that *in all cases of kidney and ureteral stones, the urinary tracts of both sides,* including the bladder, must be investigated.

Operative attack upon ureteral calculi is demanded in all cases in which the radiographic findings indicate that spontaneous delivery of the stone is improbable and when occlusion of the ureter is manifest. Of course, the latter is especially true when both ureters are occluded. In this event, the calculus is removed from one side, and in a few days, the opposite side is attacked.

The *technic* is taken up in connection with Operations upon the Kidneys and Ureters, p. 2326.

FIG. 996.— SECTION OF A TUBERCULOUS KIDNEY (Symmers, Bellevue Hospital case).

TUBERCULOSIS OF THE KIDNEY.— The surgery of kidney tuberculosis is of comparatively recent development. This is perhaps attributable to the fact that the disease was for a long time regarded as due to an ascending infection and the patient as doomed to die of "urogenital tuberculosis." The foundation of our modern conception of the affliction was laid by the work of Steinthal[43] (1885), who established the existence of *primary renal tuberculosis,* in the sense that it develops there first, as far as the urinary organs are concerned. Later, it was shown that even so-called primary renal tuberculosis is *secondary to similar lesion in other organs,* to which the bacillus more readily gains access (lung, skin, gastro-intestinal canal, etc.), and *is carried to the kidney* by the blood.

Hematogenous renal tuberculosis, in the vast majority of cases, begins in the medulla, in a papilla, or in the wall of a calix. TUBERCULOSIS OF THE TIP OF THE PAPILLA IS CHARACTERISTIC OF THE EARLY STAGE OF THE DISEASE (Rosenstein[44]).

The *further course* of renal tuberculosis is similar to that followed by the disease in other organs. It becomes confluent, undergoes caseation, and forms large, yellow, round areas of infiltration which, at times, cause the entire organ to undergo lobular deformation. The infiltrated areas are finally converted into cavities of varying size and outline; these ultimately perforate into the renal pelvis and calices and a tuberculous pyelonephritis is established.

In not a few instances the tuberculous process is restricted to a single pole of the kidney and the capsule becomes sclerotic. However, in this event, perforation into the perirenal fat is not uncommon.

Even in extensive destruction of kidney parenchyma, more or less of the latter survives and continues to functionate. However, the process extends to the mucosa of the ureters and finally invades that of the bladder. In the latter situation, the tuberculous invasion is at first restricted to the mucosa contiguous to the ureteral opening, and later, gradually (very gradually) infects the entire mucosa.

When a considerable quantity of tuberculous *débris* suddenly gains access to the ureter, the latter is often occluded, a state of affairs very likely to be responsible for *tuberculous uronephrosis*. However, here, too, our old enemies — the pyogenic aggressors — again find the conditions favorable to their nefarious activities and soon a tuberculous pyonephrosis is established.

A *classification* of renal tuberculosis is not of great practical importance, except, perhaps, with regard to the extent of destruction of tissue, but even this is not readily expressed in language. Tuffier[45] has attempted this as follows: pyelonephrosis tuberculosa; tuberculous infiltration; tuberculous hydronephrosis; and massive tuberculosis (*degenerescence massive du rien*).

Tuberculosis of the ureter is characterized by tuberculous infiltration and ulcerative degeneration of, first, the mucosa, and later, the muscularis. At the same time the outer layers of the ureters are much thickened, which obviates perforation of the tubular organ on the one hand, but is responsible for its adherence to its surroundings. The process is essentially one of thickening and sclerosis, rather than dilatation, except where central to a strictured area.

The *ureter is occasionally infected by extension upward from the bladder.* Purely hematogenous (so-called primary) tuberculosis of the bladder is very rare. In men genital tuberculosis is believed to extend to the urinary passages and ascend to the kidney, invading the other organs *en route*. It is not improbable that genital and renal

tuberculosis occur simultaneously and are derived from the same focal lesion, but that the latter is not dependent upon the former. However, that genital tuberculosis is likely to be secondary (by extension) to renal tuberculosis would seem to be shown by the experimental work of Koenig and Baumgarten,[46] who say that "tubercle bacilli do not swim against the stream." On the other hand, there would seem to be considerable clinical evidence that renal tuberculosis may extend to the bladder and then ascend to the sister organ. Wildbolz[47] furnishes an extended discussion of the question, together with the complete literature on the subject.

One may say, that the first tuberculous lesion in the kidney is hematogenous. The sister organ may be infected by the first, as stated. Lymphogenous tuberculous infection of the kidney by way of the lymphatics of the ureter is possible, but not probable (Wildbolz[47]).

Tuberculous nephritis in its acute miliary form is not of surgical importance, nor is massive renal tuberculosis, when it is a part of more or less generalized chronic tuberculosis (especially the lungs), an affliction amenable to surgical attack. However, the *appearance of tubercle bacilli in the urine* of patients afflicted with pulmonary tuberculosis, *when the kidney is apparently healthy,* presents, as can be imagined, great diagnostic difficuties. The veil is somewhat lifted when the bacilli appear in the urine of only one kidney. Of course, the presence of more or less extensive bladder lesions solves the question. However, unfortunately, these occur when the renal lesion is advanced. In doubtful cases the problem must be given careful consideration; all the measures of precision discussed above must be employed before a conclusion is permissible. This aspect of the situation has been given especial attention by Wildbolz.[47]

The *question of the soundness of the sister organ* is exceedingly important. Most of all, secondary infection of the opposite kidney, as stated, is to be feared. It is a fortunate circumstance that IN THE VAST MAJORITY OF CASES RENAL TUBERCULOSIS IS UNILATERAL. Why this should be so is difficult to explain. A predisposition in this connection is indicated by the fact that the *right kidney* is more often affected than the left. It is probable that trauma, on the *locus minoris resistentia* theory, has a certain influence in this respect. The theory that a previous embolism disturbs the normal circulatory conditions in the kidney, and thus the deposit and multiplicaton of tubercle bacilli is favored, is not without experimental support (Pels-Leusden[48]). A remarkable statistical review of renal tuberculosis, based on 40,621

autopsies, is presented by Kümmel.[1] Among the many conclusions deducible from this review, two — that renal tuberculosis is embolic and not eliminative, and that it is essentially a unilateral disease — are of surgical importance.

The *symptomatology* of tuberculosis of the urinary passages, as is to be expected, does not present a stable clinical picture. As it is primarily a unilateral disease, its early symptoms deserve especial attention. Unfortunately the disease may be existent for a long time before disturbances are manifest. It is permissible to say that, as tuberculosis of the bladder is a secondary infection, its cause must be attacked, and that removal of the infected kidney is almost invariably followed by healing of the bladder lesion. The necessity for making this statement at this time lies in the fact that the FIRST SYMPTOMS OF RENAL TUBERCULOSIS CONSIST IN DISTURBANCES OF THE FUNCTION OF THE BLADDER.

Slight turbidity of urine, without tenesmus or frequency in emptying the bladder, is the earliest manifestation in this connection. If the turbidity is accidentally discovered by the patient or is observed by the physician, the problem should not be dismissed with the assumption that a simple catarrhal cystitis is being dealt with, but should lead to a thorough examination of the urine.

Kümmel[1] says "Every chronic vesical catarrh, especially in women, not caused by gonorrhea or careless catheterization, arouses the suspicion of tuberculosis and invites the employment of measures destined to determine the facts. Not alone that, but even the presence of gonorrheal infection is not determining, as this affliction is undoubtedly responsible for conditions favoring the development of tuberculosis." In many cases this stage of the disease is attended with indefinite pelvic pain, which leads the patient to the gynecologist. THE CORRECTIONAL OPERATIVE MEASURES DIRECTED TOWARD RELIEF OF CYSTITIS, PRESUMABLY DUE TO UTERINE DISPLACEMENTS, CYSTOCELE, ETC., SHOULD ALWAYS BE PRECEDED BY THE EMPLOYMENT OF DIAGNOSTIC MEASURES WHICH EXCLUDE THE CAUSATIVE INFLUENCE OF TUBERCULOSIS OF THE URINARY PASSAGES.

In the group of cases just discussed, pathological urine dominates the picture, while disturbances in its expulsion are still absent. However, in not a few cases the general condition of the patient indicates the presence of an occult exhausting lesion somewhere in the body and a diligent search may disclose the guilty kidney condition. Unfortunately, the urine from a tuberculous kidney, even a much involved one, may not present microscopical evidence of the lesion; this is also true

when distressing bladder disturbances are already established. In these cases a complete examination of urine is indicated, more especially as THE EARLIER A TUBERCULOUS KIDNEY IS REMOVED, THE BETTER IS THE OUTLOOK AS REGARDS HEALING.

In the *more advanced cases* the clinical picture is more clearly defined and is characterized by loss of flesh, pain, and bladder tenesmus, to which may be added fever, and cloudy urine containing tubercle bacilli.

The *diagnosis* of renal tuberculosis in advanced cases is not difficult. In the early stages of the disease, when no indicative symptoms are present, discovery is, in a measure, a matter of accident. *Palpation* is helpful only when the kidney has undergone extensive changes.

In these cases *recognition of the tubercle bacilli in the urine* is of the greatest diagnostic significance. It is true that a tuberculous focus may exist in the kidney and, at first, not communicate with the urinary passages, so that the bacilli are not contained in the urine. On the other hand, repeated examinations of the urine, and its vaccination into animals, makes the discovery of the presence of the bacilli certain in the vast majority of cases of tuberculosis of the urinary system. Error as regards smegma bacilli is obviated by sterile catheterization and attention to the methods of staining the specimen. Discovery of tubercle bacilli in the urine demonstrates the presence of a tuberculous lesion in the urinary tract; its location is established by the use of the *cystoscope,* and more especially the *ureterocystoscope.* Early in the disease the cystoscopic picture does not present ulcerations in the region of the ureteral opening, but as the disease progresses these make their appearance and establish the diagnosis. Repeated cystoscopic examinations reveal the gradual dissemination of the disease over the mucosa of the bladder.

The *danger of infection,* the result of ureteral examination, if properly carried out, is very slight. This is especially true when the bladder is not yet infected, when the method really possesses its greatest value. In addition to this, ureteral catheterization and the examination of the specimen of urine obtained from the sister organ is the only certain index of the functional activity of the latter and should not be dispensed with.

Despite the improved technic of the procedure, there are still cases in which *ureteral catheterization is impossible.* To overcome this, various means have been devised, i. e., cystotomy, and catheterization of the ureters through the bladder wound, simultaneous exposure of

both kidneys, etc. In this connection the Röntgenogram is of assistance. Occasionally, palpation of a thickened ureter through the rectum is of help. In these cases, Kümmel[1] removes the undoubtedy infected kidney, if the cryoscopic findings show that the total output of urine meets the functional requirements. When the latter are insufficient, he *nephrotomizes* the manifestly diseased kidney, tampons it, and, if the sister organ takes up the excretory function, removes the nephrotomized kidney.

Renal tuberculosis follows a chronic *course*, in which the organ is destroyed and the ureter and bladder are sooner or later invaded. The process often remains relatively quiescent for a time and then becomes suddenly exacerbated; thus the disease may drag on for months and years, the fatal outcome being due to invasion of both kidneys, miliary tuberculosis, amyloid degeneration, exhaustion, etc. Prolongation of life occurs in connection with complete ureteral occlusion and sclerosis of the kidney. It is important to know that PRIMARILY UNILATERAL RENAL TUBERCULOSIS DOES NOT USUALLY EXTEND TO THE SISTER ORGAN UNTIL IT HAS EXISTED FOR A LONG TIME. This is a strong argument for early operative attack.

The *treatment* of renal tuberculosis may be regarded as surgical. It is true that *spontaneous healing* of the process occasionally takes place; this is shown by autopsy, but in these cases (and they are rare) the organ is destroyed. The healed organ always leaves a much damaged bladder, which never ipmroves to the extent observed sequential to nephrectomy.

The *dietetic, climatic,* and *medicinal treatment* of the disease, together with the use of tuberculin, is not satisfactory. Its employment must be controlled by repeated examinations, destined to inform the surgeon as to the behavior of the disease. Experience would indicate that the process is little, if at all, influenced by these measures. It is proper to say, however, that recoveries under this form of treatment have been reported (Wildbolz[47]).

Operative effort at relief in renal tuberculosis is limited to *nephrectomy.* This is only difficult when the ureter is also invaded, especially if the process extends to the bladder. Most surgeons have abandoned simultaneous nephrectomy and ureterectomy. This is justified on the ground that healing is accomplished by other, less radical, means. The subsequent tuberculous infection of the operative wound is avoided by isolation of the ureter, which is also followed by healing of the latter. After the kidney is removed, the ureter, held in a clamp,

is isolated from its surroundings with the finger and delivered through a small stab wound and sutured to the skin. This is followed by the application of pure carbolic acid to the mucosa of the ureter; healing promptly follows.

Paranephritis.— Inflammation of the perirenal fatty tissue was recognized as a separate disease by Rayer (1839). Although there has been some confusion as to terminology, today the term *perinephritis* is used when only the fibrous capsule of the kidney is inflamed.

The paranephritic tissues are invaded by contiguous extension or

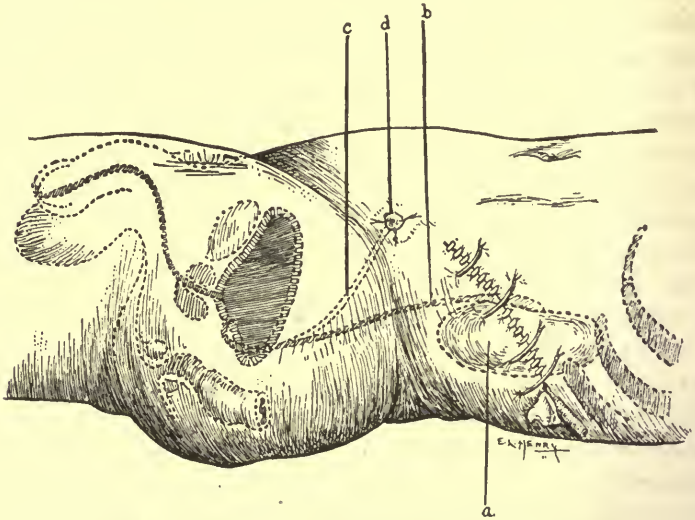

Fig. 997.— Suture of a Tuberculous Ureteral Stump to the Abdominal Wall, Distal to the Nephrectomy Wound.

a, Original location of kidney; *b*, original course of ureter; *c*, course of the ureter following the nephrectomy; *d*, ureteral stump implanted in the skin.

may be infected by processes originating at a distance from them. Thus, paranephritic suppuration develops in connection with infective processes occurring in the stomach and gut, the liver and biliary passages, and from a psoas abscess, etc., etc. All affections in the abdominal regions which are directly or indirectly responsible for infection of the retroperitoneal tissues — parametritis, permetritis, perityphlitis, oöphoritis, affections of the bladder, the testicles, seminal passages, etc. — may be responsible for paranephritis. Also, thoracic empyema may perforate the diaphragm and reach the perirenal tissues, just as retroperitoneal and subphrenic abscesses may reverse

the conditions. In many instances the diaphragm dips into the fat close to the upper pole of the kidney (Tuffier[45]).

Heretofere, the development of paranephritic suppuration in connection with acute infectious diseases (variola, typhoid, scarlatina, etc.) and comparatively slight trauma (including muscular exertion) was difficult to understand. Recently, however, the fact that the kidney is actively engaged in the elimination of pathogenic bacteria and the realization of the important rôle that enteric bacterial flora plays in the causation of all kinds of inflammatory processes, and farther, the study of the anatomical paths that infection follows have robbed the development of suppurative paranephritis of much of its previous mystery.

The anatomical considerations have been minutely studied by a number of observers (Lejars, Tuffier, Stahr, etc. See bibliography of Tuffier[45]), the result of which shows that the blood and lymphatic vessels of the fatty capsule communicate freely with those of the kidney and that extension of infection by contiguity is very likely to happen. In addition to this, it has also been shown that PATHOGENIC, INFLAMMATORY BACTERIAL EXCITANTS, WHICH HAVE GAINED ACCESS TO THE BLOOD, MAY TRANSMIGRATE THE KIDNEY WITHOUT THE SLIGHTEST EVIDENCE OF THEIR PRESENCE IN THE URINE AS FAR AS CHANGES IN THE LATTER ARE CONCERNED. This may be regarded as indicative of how readily bacteria in the capillaries of the kidney gain access to the paranephritic fatty capsule, instead of passing off with the urine.

While occasionally infection of the paranephritic tissue assumes a chronic form (usually in consort with renal lithiasis, etc.) and is attended with sclerosis of the fatty capsule, it more commonly produces suppuration and presents itself as a *paranephritic abscess.*

It, of course, goes without saying, that paranephritic abscess is frequently the result of suppurative nephritis in which a renal abscess perforates externally. This class of paranephritic abscess is secondary to either primary cortical abscess, or develops from rupture of a focus located more deeply in the parenchyma of the kidney. However, as is apparent from what has been stated above, paranephritic abscesses may occur when no renal lesion is demonstrable and the urine is perfectly normal. It would seem that traumatized paranephritic fat is exceedingly susceptible to the effect of bacteria which transmigrate the kidney tissue and produce no pathological changes *en route*, reserving this activity for the injured tissues. No doubt the occurrence of *metastatic paranephritic abscesses,* occurring in connection with acute

infectious diseases, is susceptible of the same explanation, as are also the abscesses developing in cases of furunculosis, panaris, etc. In the latter class of cases the staphylococci are delivered in the pararenal fat in the same way as occurs in connection with suppurative osteomyelitis. All of which means that the so-called "idiopathic" paranephritic abscess is no longer shrouded in darkness, but that its pathogenesis is as clearly recognized as is that of the abscess by contiguity.

Of the various *directions in which a paranephritic abscess ruptures,* the external is, fortunately, the most frequent, the exudate usually coming to the surface just above the ilium or below the twelfth rib, Occasionally an abscess ruptures into the pleura, the lung, the abdominal cavity, the duodenum, the ascending colon, or downward into the bladder or vagina.

The *clinical picture* of paranephritis is at first that of a deep seated phlegmonous suppuration with the usual attendant constitutional disturbances. Pain in the lumbar region is the rule, but is not always present; tenderness is most marked at the outer border of the sacrolumbalis muscle. If the region of the upper pole of the kidney is primarily invaded, the symptoms are likely to resemble those of pleuritis or pneumonitis, while invasion of the area of the lower pole is attended with contraction of the psoas muscle, and pain radiating into the thigh, scrotum, and vulva.

The *diagnosis,* at first, is likely to be difficult, the affection being often mistaken for the beginning of an acute infectious disease. During this stage a conclusion is not possible.

When the abscess is developed the diagnosis becomes patent. It is then based on swelling in the region below the ribs, which is especially noticeable when the patient assumes the sitting position. The tumor is sharply outlined, immovable and, in advanced cases, the superimposed skin is edematous. *Fluctuation* is discernible late in the process. In doubtful cases *aspiration* (posteriorly) is permissible.

The *treatment* consists in wide incision of the abscess through the lumbar region, employing the oblique method of approach, as for nephrectomy. In chronic paranephritis the kidney is exposed as for nephrectomy and the thickened paranephritic tissue is excised. This is a very gratifying procedure; to prevent subsequent prolapse of the kidney, it is sutured to the muscle, as in nephropexy.

Nephritis (Essential Hematuria, Nephralgia).— While most textbooks, especially the older ones, contain a chapter devoted to essential renal bleeding (renal hemophilia, *neuralgie hématurique,* renal

epistaxis) and renal neuralgia, it has recently come to light that in most instances the picture characterized in this manner is produced by one of the various forms of nephritis. Nevertheless it is clear that essential *bleeding,* without other symptoms, still exists as a clinical entity (Albarran[49]).

Renal hemorrhage, as such, may be classified as follows:

1. Bleeding following great and prolonged exertion, such as horseback riding.
2. Periodic bleeding in children, occurring in connection with rachitis, etc.
3. Bleeding in connection with movable kidney, presumably due to congestion.
4. Bleeding during pregnancy and lactation, also due to congestion.
5. Profuse bleeding in which the only lesion consists in areas of sclerosis of the kidney.
6. Bleeding due to relatively slight trauma.
7. Genuine renal hemophilia.
8. Bleeding due to telangiectasis of the vessels of the renal pelvis (very rare).
9. Hysterical bleeding, which occurs after a violent emotion (very rare).

In the *diagnosis* it is of course necessary to exclude all other causative factors. Difficult as this may seem, a careful study of the facts, together with close scrutiny of the catheterized specimen, will lead to a correct conclusion, except, perhaps, in early tuberculosis and beginning neoplasm.

Kidney pain, i. e., renal neuralgia, has, as the outcome of refinements in diagnostic methods, become a rare condition. It is probably always due to an organic kidney lesion.

Nephritis.— For a long time, not long gone into history, operative attack upon the kidney was limited to cases presenting renal pain, and bloody and turbid urine, presumably caused by stone, tumor, tuberculosis, etc. The fact that exposure of the kidney did not uncover the presence of these conditions led to the conclusion that the operation, with its coincident exploratory nephrotomy, had been unnecessarily employed, until the surgeon was confronted with the surprising realization that this seemingly unwarranted maneuver was followed by disappearance of the disturbances and complete recovery of the patient. In cases in which radicalism led to nephrectomy, microscopical exami-

nation of the extirpated kidney showed that the changes responsible for the disturbances consisted in some form of nephritis.

The outcome of prolonged investigation has made it permissible to divide these changes into two groups, viz., *acute* (or subacute), and *chronic* nephritis.

Acute nephritis, considered from the standpoint of the surgeon as amenable to operative measures of relief, may be presented as follows:

a. *Acute nephritis developing in connection with acute infectious diseases* (especially scarlatina) was first attacked by Harrison (1896), who incised the capsule of the kidney on the theory that the measure relieved intrarenal tension in the same way as iridectomy influenced the conditions in the eyeball in glaucoma. Harrison characterized the kidney condition as a "pernicious kidney glaucoma" and held the increased pressure responsible for anuria, a view borne out by subsequent observation.

b. *Toxic nephritis,* due to the irritating effect of *chemical poisons,* and characterized by *anuria.* The latter is always fatal unless the intrarenal pressure is relieved by decapsulation of the kidneys.

Eclampsia belongs in this category. Ninety-eight cases of decapsulation of the kidneys in eclamptic aunria are anaylzed by Poten;[50] 60 recovered.

c. *Acute infectious nephritis* (nephritis apostematosa) is, to the surgeon, the most important form of the afflictions of this class. The lesion consists in *acute suppurative nephritis with miliary abscesses.* In most cases a pyelitis extends from the pelvis to the calices, so that, finally, the entire kidney is converted into a single abscess cavity, while in suppurative nephritis the kidney is studded with multiple miliary abscesses, i. e., the kidney parenchyma of itself is infected.

The *characteristic symptom* of suppurative nephritis is pain in the region of the kidney; this is often attended with increased muscular tension in the same situation (*defense musculaire*) and may be as indicative as it is in connection with appendicitis with perforation. To this is added the picture of an acute infectious disease.

The *urine* is turbid and contains leukocytes and bacteria, provided the kidney is capable of excretion; it often emits the characteristic odor of colon infection and, usually, also contains red blood corpuscles and hyalin casts. As the disease is frequently *unilateral,* ureteral catheterization is essential. In *bilateral* infection anuria is likely to occur.

Spontaneous healing of suppurative nephritis is possible; in this

event, the miliary abscesses coalesce and form a single abscess cavity, which ruptures into the pelvis, or perforates the capsule, forms a paranephritic abscess and ruptures externally. However, this is not to be expected; the indication is to expose the kidney at once and to nephrotomize it. The measure is followed by extremely satisfactory results. If the infected kidney parenchyma is destroyed, the organ must be removed.

Chronic nephritis, as a surgical problem, may be arranged in three groups:

a. *Nephritis dolorosa* is characterized by *pain* and was previously classed as renal neuralgia. The pain in the class of cases under consideration cannot be connected with lithiasis or other pathological causative factors discoverable upon splitting the kidney. However, later and more careful investigations showed that the exposed kidney was *covered by a much thickened, shrunken capsula propria* adherent to the perirenal fat and continuous with insular sclerotic foci, which dipped into the renal parenchyma and were surrounded by zones of hyperemia and stasis. In short, the process represents circumscribed areas of beginning nephritis. In addition to this, careful examinations of specimens of urine obtained by ureteral catheterization revealed the presence of casts, and often small quantities of albumin. Despite this apparently clear picture, it is not always possible to be certain whether the kidney pain is due to the condition stated or to the presence of small stones which escape recognition. The *diagnosis* of nephritis dolorosa, of necessity, is only permissible when the pain and other symptoms are limited to one side.

b. *Hemorrhagic nephritis*, i. e., chronic nephritis with bleeding not ascribable to the causes stated and attended with nephralgia, also not due to recognized causative factors, presents a clinical entity difficult to recognize as such.

The *diagnosis* must *perforce* rest on a careful examination of specimens of urine obtained by ureteral catheterization. Patients afflicted in this way are likely to present a cachectic appearance which, together with the presence of blood in the urine and the absence of a bladder lesion, is equally indicative of beginning new growth in the kidney.

The *treatment* in renal bleeding consists in its arrest. As the diagnosis is uncertain, the indication is to expose the kidney and then decide the technic of the measure of relief. *Nephrectomy* is justified only when the hemorrhage is violent. Decapsulation accomplishes the

purpose in most instances; it has been followed by clinical healing in a sufficient number of cases to justify its employment with the restrictions mentioned above (Kümmel[1]).

c. Morbus Brightii, i. e., kidney disease in which bleeding is not a dominant factor, but in which the lesion consists of a typical *chronic parenchymatous and interstitial nephritis,* may also be included in the category of surgical diseases.

Decapsulation.— So-called "medical nephritis" (which is always bilateral) was considered as belonging entirely to the domain of internal medicine until Edebohls[51] demonstrated that the fixation of a nephritic kidney was followed by improvement of the renal condition. As the improvement was presumably ascribable to the release of renal tension, logically the next step was to decapsulate the nephritic kidney. In the following decennium a number of experimental and clinical observations were made, which seemed to show that the benefit derived from the method was due to the establishment of a new (collateral) circulation. While the method did not accomplish what was at first hoped for, its employment certainly prolongs life and, in some instances, is followed by symptomatic recovery. It is, however, in cases of oliguria and anuria in connection with chronic nephritis, that the measure has attained its most brilliant results. It is perhaps best to execute the maneuver under local anesthesia. The technic of the procedure is simple: The kidney is exposed as for nephrectomy, the capsule split along the convex border of the kidney, separated on either side as far as the hilus, and cut away. The wound is closed without drainage (de Bovis,[52] Casper,[53] LeDentu,[54] Ferguson,[55] Guiteras,[56] Kümmel,[57] Rovsing,[58] Tuffier,[59] Zondek[60]).

Renal Lues.— Lues appears in the kidneys in the form of *gumma* and is a *diffuse* infiltrate or an *amyloid* degeneration.

Gummata form in the renal cortex or in the pyramids. They originate in the interstitial tissue, compressing the urinary tubules; microscopically they bear some resemblance to tuberculosis.

Diffuse luetic nephritis appears, at first, as a large white kidney, and later the organ undergoes sclerosis. The process may be unilateral.

Neither form of the disease possesses especial surgical interest. The affliction presents the *clinical picture* of chronic nephritis without pain, and is usually associated with luetic hepatitis. Patients afflicted with luetic nephritis do not bear mercurial treatment well, the damaged kidney being unable to eliminate the drug, and poisoning or

even mercurial anuria may develop with moderate dosage of the agent. De Grandmaison[61] presents a comprehensive study of renal lues.

Tumors of the Kidney and Pelvis.— The kidney, with its immediate surroundings, is not rarely the seat of new growths of various kinds, a part of which originate in the tissues of the kidney or its pelvis, while others arise in the fatty capsule or the suprarenal gland. In addition to this, embryonic rests of the wolffian body are no doubt responsible for a number of tumors developing in this situation. Finally, the kidney is not infrequently the seat of cystic parasitic tumors (echinococci) and of retention cysts.

A purely histological *classification* of kidney tumors is not difficult. On this basis they may be divided into those arising from preformed *connective tissue;* from preformed *epithelial cells,* and from *heterogeneous embryonic elements,* and those developing in the form of *retention* or *parasitis* cysts. These may be arranged into groups as follows:

1. Fibromata and lipomata, osteomata and enchondromata, angiomata and lymphangiomata, sarcomata, angiosarcomata, endotheliomata and peritheliomata, and their mixed forms.
2. Adenomata, cystadenomata, adenocarcinomata, and carcinomata.
3. Hypernephromata and embryonic glandular tumors of childhood, including rhabdomyomata.
4. Polycystic renal degeneration and solitary renal cysts.
5. Renal echinococcus.

From a surgical standpoint, however, this grouping is of little practical value and it is therefore preferable to arrange the subject into *benign* and *malignant* tumors, placing hypernephromata in a class by themselves and devoting separate consideration to *cystic renal tumors.*

Renal tumors that possess clinical importance appear at two distinct periods of life, first, in very early life or before puberty, when they are congenital; and second, after forty years of life; thus leaving comparatively free the years when renal tubercuIosis is most common.

The *chief landmark of tumor formation of the kidney* is, of course, the recognition of a solid renal mass. Unfortunately the hidden location of the organ makes this difficult, not alone for the patient but also for the physician, unless the tumor happens to originate at the lower pole of the organ, when it obtrudes itself into a readily palpable zone. When the tumor is palpable, it must be interpreted from the standpoint already taken up in connection with tumors in general (Part, VI, chap. iii).

As will be shown later, the appearance of *hematuria* is usually the first subjective symptom of renal tumor, and, according to Chevalier,[62] it occurs in 26 per cent of the cases. The signs indicative of the renal origin of the bleeding have already been much discussed above. If the growth of the tumor produces extensive intrarenal bleeding, the coincident increase of tension is responsible for the same symptom complex presented when the tension is due to other causes. In a certain number of cases the blood is discoverable only by microscopical examination (Israel[63]). The frequency of renal colic provoked by the bleeding of renal tumors has led to the employment of the term "neuralgic form of renal tumor," used by the French urologists. A certain diagnostic value is attached to the appearance of varicocele. Disappearance of the latter has been noted after removal of the tumorous kidney (Heresco[64]).

The *extension of renal tumors* depends upon their character. The kidney is not a law unto itself in this regard. Carcinoma extends by the lymphatic and sarcoma by the blood vessels, or the tumor invades the contiguous organs by direct extension. The motility of the tumorous kidney is of diagnostic value in this regard.

In all cases of tumor of the kidney the functional activity of the sister organ must be investigated.

The *therapy* of malignant tumors of the kidney consists in nephrectomy. Resection of the kidney for the relief of a kidney tumor of any kind is of questionable utility.

Of the *benign tumors* of the kidney, angiomata, lymphangiomata, osteomata and enchondromata rarely attain large dimensions, and possess little, if any, clinical significance. On the other hand, lipomata, fibromata, adenomata, and their mixed forms often assume gigantic proportions and must be removed.

Primary cancer of the kidney presents itself, macroscopically, in two forms — the *nodular* and the *infiltrating*. In this regard the kidney does not stand alone, nor is it any exception with regard to the general law as to the relative malignancy of medullary and scirrhous carcinomata.

Waldeyer[65] has shown that renal carcinoma originates in the preformed glandular anlage of the urinary tubules. Manasse[66] and Sudek[67] seem to have shown that the primary nodular form belongs to the adenocarcinoma class, and that the infiltrating form originates in the epithelial elements of the urinary tubules. The entire problem is analyzed by Graupner.[68]

Clinically, renal carcinoma does not present a clearly defined picture; it will rarely be possible to go farther than the assumption that a malignant process is being dealt with. This assumption is justified when a solid tumor is palpable in the kidney region and this is attended with hematuria and the urine contains tumor elements.

Simple sarcomata of the kidney do not present histological peculiarities; they usually belong to the spindle, giant, or small round cell varieties. Of course mixed tubors, i. e., fibro-myxo- and chondro-sarcomata, do not fail to make their appearance; also, of course, the

FIG. 998.— PRIMARY CARCINOMA OF THE KIDNEY.

tumors vary widely as regards malignancy. Hemorrhages and regressive metamorphoses are very common. Hematuria occurs in fifty per cent of the cases (Chevalier[62]).

Angiomata, endotheliomata, and peritheliomata of the kidney represent a group of tumors originating in the endothelial and perithelial cellular elements of the blood and lymph vessels. The perithelial cells have large dark nuclei and contain numerous protoplasmis bodies, which possess fine prolongations and lie outside of the blood vessels (Waldeyer[65]).

With regard to the exact pathology of this class of tumors, Küm-

mel[1] presents an analysis of the various views of those who have studied the problem; this cannot be reproduced here. Kümmel accepts the conclusion of Manasse,[66] who divides angiosarcomata into *blood vessel endotheliomata, lymph vessel endotheliomata,* and *perivascular sarcomata.* The last he regards as identical with *peritheliomata,* and considers the latter term justifiable if one accepts the name perithelial as properly applied to the cells of the adventitia described by Eberth (see Manasse[66]). The characteristics of this group of tumors are discussed in Part VI, chap. xii.

Embryonic sarcomatous glandular tumors (mixed tumors) *of childhood* (embryonic adenosarcomata) present certain peculiarities. While so-called hypernephromata originate from displaced congenital embryonic renal tissue, but usually do not make their appearance until late in life, there are other forms of renal tumors, also the outcome of errors in development, that grow with great rapidity, are exceedingly malignant in character, and are found in the newborn, or make their appearance in the first few years of life. These growths have been described as *sarcomata, carcinomata,* and as *sarcomata with striated muscle fibers* (*rhabdomyosarcomata*), although not any one type was clearly defined. On the other hand, it would seem that just this mixture of anatomical elements, each in its embryonic type, constitutes the chief characteristic of the tumor under consideration. The quantitative contribution of the glandular or connective tissue elements may vary widely, and when these in turn are carefully scrutinized, their particular structure may also be widely divergent, one microscopical section showing adenoma or carcinoma and another spindle cell sarcoma, etc.

It is patent that authors, in describing a given tumor of this sort, were prompted to apply to it a designation in accord with the domination of certain kinds of tissues, arbitrarily employing the term carcinoma, adenocarcinoma, sarcomatous carcinoma, adenosarcoma, adenochondrosarcoma, or myxosarcoma. As this is misleading, one is justified in grouping these malignant growths together and designating them as *embryonic or sarcomatous glandular tumors of the kidney region of childhood.* The astonishing peculiarity of this class of tumors is that its epithelial elements do not penetrate into neighboring tissues in the manner characteristic of carcinoma cells; epithelial and sarcomatous elements proliferate together, each retaining its embryonic character. Wilms[69] explains this on the ground that new embryonic tissue is developed which always repeats its original form. The con-

nection that these tumors are believed to have with the development of the fertilized ovum is taken up in Part VI, chap. xii.

Clinically, the "embryonic glandular tumors" form a characteristic group sharply defined against malignant hypernephromata. The latter are restricted to adult life (usually after forty), the former appear early in childhood. Hypernephroma grows slowly, the glandular tumor of childhood with great rapidity. Hypernephroma is frequently attended with hematuria; in the other growth it is absent. The child afflicted with an embryonic glandular tumor of the kidney usually comes under observation when the growth has attained gigantic proportions and the chances of its successful removal are slight. As the process does not form metastases, early extirpation of the tumor is likely to be followed by permanent healing.

Hypernephroma (epinephroid, strumae suprarenales abberantes). — The causative relationship between displaced adrenal embryonic tissue and tumors lying beneath the tunica albuginea was established by Grawitz[70] (1884). Later, it was shown that these elements are responsible for a group of tumors which are by no means always benign, but, on the contrary, often become sarcomatous or carcinomatous in character. A study of the very extensive literature devoted to the subject would seem to indicate that a conclusion with regard to the exact nature of these tumorous formations is not permissible at this time (Burkhardt[71]). It would appear that genuine kidney tumors may develop within displaced adrenal tissue, and that kidney tumors developing in connection with adrenal tissue need not originate from it.

The tumors arising from adrenal tissue may be arranged in the following forms:

1. The small so-called lipomata, consisting of adrenal tissue alone and including a zona pigmentosa, are located in the cortex of the kidney. They are probably simple adrenal glands.

2. The true type of struma accessoria, which lies beneath the kidney capsule, most often appears at the upper pole, and is divided into lobules by vitreous connective tissue. The tumors crowd into the kidney tissue, are surrounded by a connective tissue capsule, and are benign in character.

3. Another group of tumors belonging to this class is characterized by active proliferation not alone of its cell complex, but also of the individual cells, which increase in number and size. The cellular deformation may make it difficult to recognize their character.

4. A rare form of tumor which consists in *pure hyperplasia of the*

cells of the pararenal gland and is also found in the cortex of the kidney (Manasse[66]).

5. A type of tumor in which not only the connective tissue spaces are filled with solid cell cylinders, but also contain hollow spaces (tubular or cystic) which are lined with proliferated epithelium. These were for a time believed to originate from the kidney tubules, but have recently been shown to develop in displaced adrenal tissue (Manasse[66]).

6. It would seem that the changes (tubular and cystic) in an adrenal tumor stated under group 5 heralds the beginning of *malignant changes* (Israel[63]). On the other hand, there is reason to believe that the malignancy is not always dependent upon the development of these hollow epithelium lined spaces, but that a sudden proliferation of connective tissue elements may occur and the process then takes on the characteristics of *sarcoma.* In other words, the histological picture of malignant hypernephroma, despite the epithelial character of its cells, may be that of sarcoma.

The *clinical course* of hypernephromata is usually a protracted one. The tumors grow slowly and do not cause disturbances for a long time. They usually present themselves after the fortieth year of life and are announced by an attack of hematuria, frequently attended with kidney colic. Even after this primary signal, advance of the growth is likely to be hesitant. Not infrequently, five, eight, ten and even fifteen years elapse between the first symptom and the final nephrectomy.

This indolence, however, does not always obtain; one can never be certain when malignant changes occur, so that recognition of a condition of affairs indicative of tumor formation should be met by operative efforts at relief.

Cystic Tumors of the Kidney.— Aside from the development of cystic spaces in malignant tumors, cysts occur in the kidney in connection with *echinococcus* and with a peculiar *cystic degeneration* of the kidney itself.

Echinococcus cysts of the kidney are not common; they grow slowly and are not likely to be responsible for disturbances. They are usually discovered in the course of operative exposure of the kidney.

When the process is uncovered, it is not uncommon to find sufficient normal kidney substance to warrant suture of the cyst wall to the wound; this is likely to be followed by obliteration of the sac (Kümmel[1]).

Cysts developing from the kidney itself are found in two forms. The more menacing is that of *polycystic degeneration* of the kidney; the other form is harmless and consists in the *solitary cyst.*

Polycystic degeneration is in part congenital and in part developed, like other cysts of the kidney, late in life. There is no doubt that more than one pathological process is responsible for cystic degeneration of the renal parenchyma. Although the macroscopical appearance of these cysts bears a close resemblance, one to another, they are, on close scrutiny, found to be variously formed and due to widely different anatomical changes.

A number of theories designed to explain the character of these forms of kidney degeneration have been advanced. The investigations made in this connection are summarized by Nauwerck and Hufschmidt,[72] who conclude that in *one form* of the disease the process is analogous in the newborn and in the adult, consisting in both instances in *connective tissue proliferation.* This extends from the ureter and pelvis to the calices and medullary tissues, and strangulates the urinary tubules, thus leading to the formation of *simple retention cysts.* In the adult the process never develops to the extent that it does in the newborn.

In the other form of cystic kidney the process is ascribable to proliferation of the epithelial calls of the urinary tubules, which finally undergo granular and colloid degeneration and convert the tubules into cysts of varying dimensions. In some instances papillary outgrowths obtrude into the cyst cavities, as in papillary cyst adenoma of the breast (p. 1871). Contiguous cysts may be separated by septa or may communicate with one another. As a rule, the cysts are filled with exfoliated epithelial detritus. These changes are not attended with active connective tissue proliferation, and in this may be regarded as quite distinct from those occurring in the first form of polycystic kidney.

Clinically, the congenital polycystic kidney does not possess great importance; as it is usually bilateral, the child does not survive. In the rare cases in which the process is unilateral, its extirpation is indicated. Even the late acquired form of the disease is often bilateral and the outlook is unfavorable.

Removal of unilateral acquired polycystic kidney in the adult has not infrequently been accomplished; the mortality is high. On the other hand, if one could be sure of the form of the process uncovered

through the wound of approach, an effort to accomplish healing by suture of the cystic organ to the skin would be justified.

Solitary cysts of the kidney are very rare and are usually found at its lower pole. They originate in the cortex of the kidney and are regarded as retention cysts of the glomeruli or the urinary tubules. Occasionally a cyst of this sort attains great size (adult head); they are easily removed by resection (Viguard[73]).

FIG. 999.— POLYCYSTIC KIDNEY OF THE EPITHELIAL PROLIFERATIVE TYPE (Symmers, Bellevue Hospital case).

Primary new growths of the renal pelvis have been studied by Albarran,[74] and are uncommon. The lesions originate, of course, in both the epithelial and connective tissue. Of seven cases collected by Albarran, four were rhabdomyomata; one myxoma; one angiosarcoma, and one lymphatic endothelioma.

The character of these processes and their location would naturally provoke symptoms of obstruction to the urinary flow and hematuria.

One can conceive that the *diagnosis* might be made if a fragment of tumor were accidentally removed with the ureteral catheter and the surgeon were gifted with an unusual imagination. Nash[75] contributes an interesting communication on the subject of villous tumors of the renal pelvis.

Aneurisms of the renal artery at the hilum may be responsible for

fatal hematuria. The violence of the bleeding would lead to exposure of the kidney, and thus the cause of the bleeding would be uncovered. Keen[76] has collected seven cases of aneurism of the artery outside the kidney. Most of these were operated upon for large tumors; pulsation was absent in all of them. Nephrectomy was followed by recovery.

Diseases of the Ureters.— Inflammation of the ureter (ureteritis) is rarely an independent disease; it is usually secondary either to ascending (from the bladder) or descending (from the kidney) inflammatory processes. It is a curious fact that the mucosa of the ureter possesses a relative immunity against most of the infections, but not to those of gonorrhea and tuberculosis. The former promptly leads to invasion of the renal pelvis and is attended with grave changes in the ureteral mucosa, which in turn are followed by stricture formation, etc., while tuberculosis of the ureteral mucosa is responsible for great swelling, ulceration, massive proliferation of the submucous connective tissue, and, finally, the entire ureter is converted into a rigid, inelastic, enormously thickened tube. However, when once pyogenous infection of the kidney is firmly established, the ureter is correspondingly invaded.

Although *isolated inflammation* of the ureter is rare, it undoubtedly occurs. The condition is recognized as such only during operative exposure. Not a few cases are reported in the literature (Reynolds[77]).

Strictures of the ureter are the outcome of a number of conditions; they are classified as congenital and acquired.

Congenital strictures of the ureter are located at its upper and lower ends and are frequently responsible for uronephrosis in the newborn.

Acquired strictures of the ureter may be due to trauma or infection. To the first group belong postoperative strictures and those due to pressure by calculi; the second form is the outcome of proliferation of connective tissue occurring in connection with inflammatory processes.

The *symptoms* are those of renal colic. The *treatment* is taken up in connection with operations on the kidney and ureter.

A peculiar and not very rare condition of the ureters is described under the term *ureteritis cystica*, and is characterized by a cystic invasion of both ureters and the trigone. The cysts consist of a connective tissue wall lined by a layer of pavement epithelium; these are regarded as dilated glandular elements (Marckwald[78]).

Tumors of the ureter are usually secondary to those of the kidney. Primary growths are likely to be villous papillomata (Le Dentu[79]).

A discussion of the *diagnosis* of the diseases of the ureters would not

accomplish much. As a matter of fact, their consequences are chiefly of interest to the surgeon. That is to say, they usually lead to hydronephrosis or other lesions of the kidney and are uncovered during operative efforts at relief directed toward this organ. Their relationship to lesions of the kidney must be borne in mind during the operation. Cystic dilatation of the lower end of the ureter must be given consideration in connection with the bladder.

Operation upon the Kidneys and Ureters.— As the kidney is essentially concerned in elimination and this is, of necessity, more or less interfered with as the result of renal disease, an adequate *preparation of the patient* preliminary to the employment of operative measures is of great importance. Especial care is taken to regulate the heart's action, and the vicarious organs of elimination — the skin and gastro-intestinal tract — are employed for the purpose of lessening as much as is possible the burden of the kidneys in this connection. An effort must also be made to decrease the bacterial flora of infected urine by the administration of urinary antiseptics and the ingestion of large quantities of water.

On general principles the avoidance of narcosis is desirable; local or spinal anesthesia may be employed with the restrictions mentioned in Part I, chap. xii.

After the kidney is exposed the several methods of procedure as indicated by the condition presented may be stated as follows:

1. *Decapsulation,* which consists in splitting and resection of the capsule of the kidney.
2. *Nephropexy,* the operative fixation of the movable kidney.
3. *Nephrostomy,* the establishment of a renal fistula for various therapeutic indications.
4. Operative treatment for *injuries of the kidney.*
5. Operative separation of *horseshoe kidney.*
6. The operations for the relief of *paranephritis.*

These operative measures are taken up with the various conditions already discussed. This leaves for consideration, *nephrectomy, nephrotomy,* and *resection.*

The kidney is exposed in two ways: by the lumbar and the transperitoneal routes.

Nephrectomy; Extirpation of the Kidney — Lumbar Nephrectomy.— In all forms of lumbar incisions the *position of the patient* is arranged so that he lies on the sound side, slightly inclined forward, with a thick roll placed between the dependent costal arch and the

pelvis, so that the body is curved and the costo-iliac space on the upper side is increased in length. Most operating tables are constructed so that this position is readily attainable (p. 115).

Of the various *incisions* employed for the purpose of exposing the kidney, no other possesses the advantages of the one here described.

The *v. Bergmann-Israel incision,* as modified by Kümmel,[1] makes simultaneous exposure of the ureters possible. It begins at the anterior edge of the sacrolumbalis muscle, about 1 cm. below the twelfth rib and extends forward and obliquely downward, following the course of the ureter. Its length varies with the problem at hand. The muscles and the transversalis fascia are divided, exposing the retroperitoneal fat. If the exposure is insufficient, the incision is prolonged downward; the space thus gained is usually sufficient for all practical purposes. If the upper pole of the kidney and the infra-diaphragmatic space beneath it are inaccessible, it may be necessary to resect the twelfth, or even the eleventh, rib. The latter step is employed only in exceptional cases; involving as it does the danger of injury to the pleura, it should not be lightly undertaken.

Care should be taken to avoid injury to the *peritoneum* and colon. As a rule, the *peritoneum* is displaced with little difficulty. However, opening this membrane is not a calamity, if the colon is spared. As a matter of fact, the great advantage of the lumbar incision is that it permits of resection of portions of the peritoneum and its subsequent repair with little danger of serious consequences.

While an assistant holds the costal arch upward with retractors, the kidney is gradually delivered through the wound. Occasionally satisfactory *ligature of the vessels of the hilus* cannot be accomplished before the kidney is extirpated. In these instances the stump is encircled with an elastic ligature and the section is made so that a small portion of kidney tissue is left *in situ.* Proper control of the vessels then becomes a simple matter and the wedge shaped residual piece of kidney is trimmed off.

The rule is to extirpate the kidney between the capsula propria and the fatty capsule. However, this is subject to variations. In cases of malignant disease and in tuberculosis, it is best to remove the fatty capsule also; the latter may have to be accomplished after the kidney is removed. When the fatty capsule, as the outcome of chronic inflammatory processes, is very intimately associated with the kidney, the latter may have to be enucleated from within its proper capsule. In

these instances it may become necessary to remove the renal parenchyma in pieces — the *morcellement* of Tuffiier.

Transperitoneal Nephrectomy.— Transperitoneal nephrectomy possesses some advantages over the lumbar method. It gives better exposure of large tumors and permits of scrutiny of the sister organ. However, the method involves much trauma to the abdominal contents and, if the renal lesion is infectious in nature, exposes the peritoneum

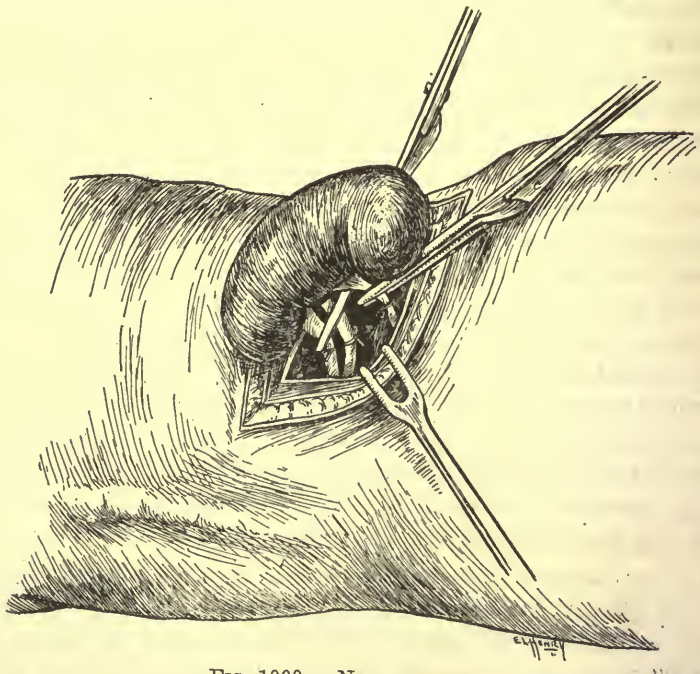

Fig. 1000.— Nephrectomy.

The kidney is delivered. The ureter and the blood vessels are clamped and ligated.

to grave dangers in this connection. The writer regards abdominal approach permissible in cases in which a *renal injury* is complicated by an abdominal one, and in cases of *pelvic kidney* (see above). The celiotomy incision is preferably made through the rectus muscle, and the kidney is exposed through the outer leaf of the mesocolon, as shown in Fig. 1001.

The *clinical consequences of extirpation of the kidney* are, as a rule, not serious. However, in a certain number of cases, the measure is

followed by the appearance, in the urine, of albumin, blood, and casts for from two to three days following the operation, and in a lesser number of instances, fatal anuria rapidly develops. The characteristic findings in these cases consist in *acute swelling of the epithelium of the looped urinary tubules,* which obstructs their lumen. Investigation would seem to show that these pathological changes are the result, on the one hand, of the narcosis, and that chloroform is more toxic in this regard than ether; and on the other, of the use of antiseptics in the wound.

The *remote effect* of nephrectomy may be stated as follows:

1. The fate of most of the nephrectomized patients is decided within the first year, during which a number die, especially those afflicted with tuberculosis, tumors, and severe pyonephrosis with or without stone.

2. If cases of renal tuberculosis or tumor survive two or three years, the outlook for the future is favorable.

3. When both kidneys are affected the remaining kidney improves if the operation is performed early.

4. In a general way, one may say, that if a patient remains well for four years, the span of life does not vary from that of the normal person.

The *anatomical changes in the remaining kidney* consist in enlargement of the glomeruli and in increase in the spaces between them, together with a more marked contortion of the convoluted tubes; a genuine hyperplasia does not occur.

Resection of the kidney consists in extirpation of the diseased sector of its parenchyma. The measure is indicated in cases of trauma, fistula, insular areas of inflammation, and benign tumors.

After the kidney is exposed the procedure is carried out in accord with the conditions presented. Benign tumors and cysts are extirpated by wedge shaped or longitudinal excision. The latter method is attended with less bleeding than the former.

NEPHROTOMY.— Nephrotomy (splitting of the kidney) is permissible in connection with a number of conditions; at times the measure is purely exploratory. However, it is employed with the view of preserving the organ, and is of distinct therapeutic value in various forms of nephritis, inflammatory lesions, multiple abscesses and uro- and pyonephrosis, in which it is not necessary to remove the organ.

After the kidney is exposed in the usual manner and delivered through the wound, the vessels at the hilum are compressed within a

suitable clamp or by the fingers of an assistant. The capsule is freely opened ½ cm. behind the greatest height of the convex border of the kidney and the latter is sectioned as deeply as is necessary to accomplish the intent; it may be opened into the hilum.

When the kidney is attacked for the purpose of removing stones (*nephrolithotomy*), the incision in the organ is made to conform to the size of the calculus. The location of the latter is determined with the probe. Using the probe as a guide, the knife makes the stone acces-

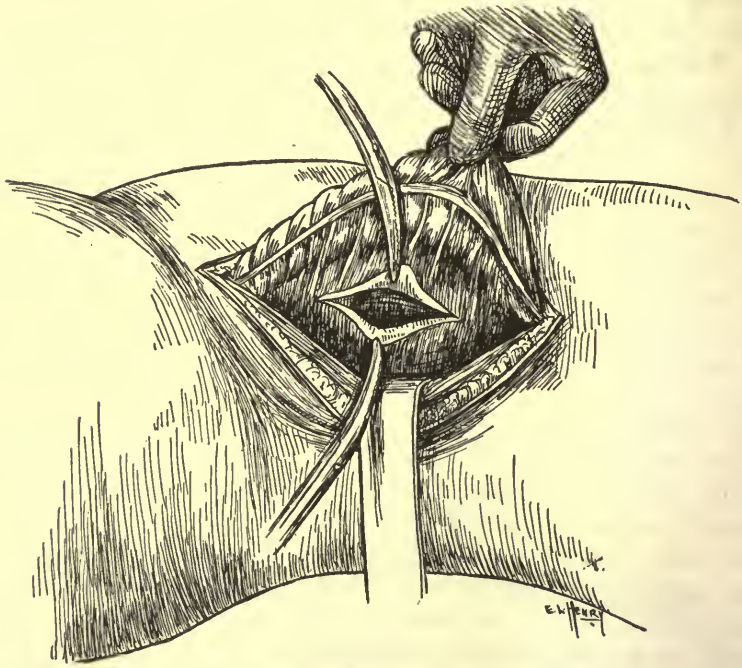

FIG. 1001.— TRANSPERITONEAL NEPHRECTOMY.

sible and it is then removed with forceps. When a large stone, together with a quantity of sand, is present in the pelvis, the kidney must be widely sectioned and all foreign material removed, using gentle lavage for the purpose. In all cases of nephrolithiasis a flexible sound is passed from the pelvis through the ureter and the patency of the latter assured. The sectioned kidney is apposed with mattress sutures, and drained. The capsule is closed with catgut.

Of the postoperative complications following nephrectomy, *renal fistula* and *secondary bleeding* are the most important.

The *correction of renal fistula* often presents considerable difficulty. If it is due to an obstructed ureter, dilatation of the latter through the ureterocystoscope may accomplish the purpose. Failing in this, if functional tests show the sister organ to be sufficient, the fistulous kidney may be removed.

Secondary bleeding is a grave problem. It is evinced by hematuria and the formation of a lumbar hematoma, which develop about the ninth day after the operation. Immediate nephrectomy and ligature

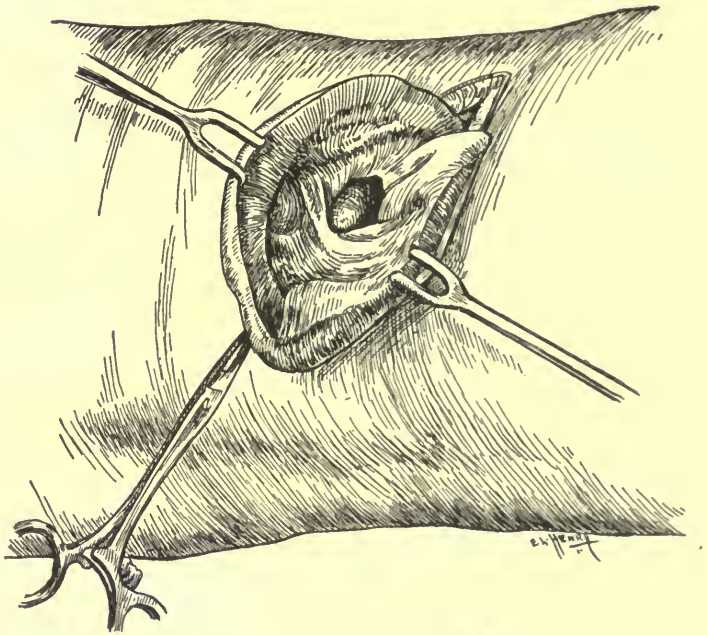

FIG. 1002.— NEPHROLITHOTOMY.

Stone in the renal pelvis exposed by nephrotomy.

of the renal vessels, as far from the hilus as is possible, must be carried out.

Operations on the Ureters.— Exposure of the ureter may be accomplished by the retroperitoneal or the transperitoneal route. The former is almost universally employed for the removal of calculi and the relief of strictures and obstructive adhesions; transperitoneal exposure is used in connection with the execution of classic operations and implantation of the ureters into the gut.

In the *extraperitoneal route* the upper half of the ureter is exposed

by a simple prolongation downward of the incision described in connection with lumbar nephrectomy. This prolongation lies two or three finger breadths to the inner side of the spine of the ilium and parallels Poupart's ligament. If the ureter must be exposed near the bladder, the incision is continued farther downward and the deep epigastric vessels are ligatured. It is to be remembered that the ureter is associated with the posterior peritoneum and when the latter is reflected the ureter goes with it. In all cases it is desirable that catheters be placed in the ureters before they are attacked. As a rule,

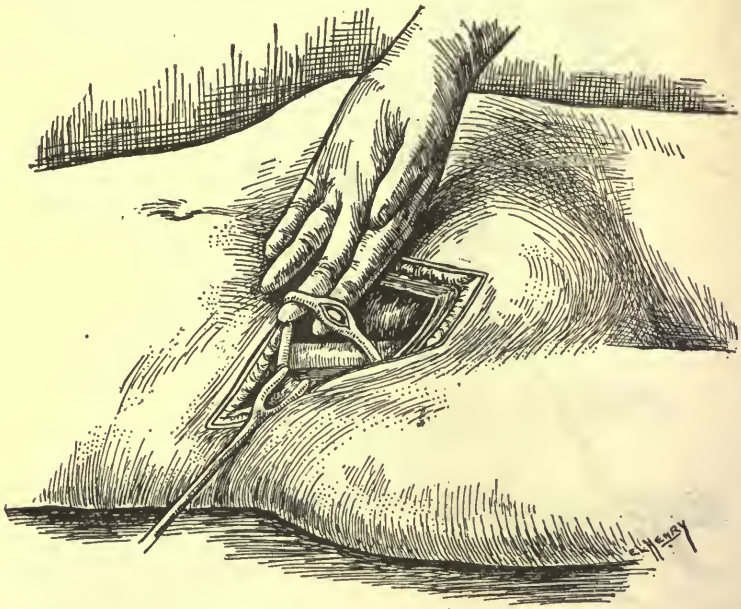

FIG. 1003.— URETEROLITHOTOMY.

The ureter is delivered and opened, exposing the stone.

identification of the ureter is not an involved problem nor is its isolation attended with great difficulties, unless the tubular organ is bound down by periureteral adhesions.

Ureterolithotomy must always be preceded by establishment of the location of the stone by the Röntgenogram, catheter, etc. The ureter is delivered into the wound and opened over the stone by a longitudinal incision (Fig. 1003), which permits extrusion of the concrement without trauma to the divided coats; at times the scoop must be used. When the stone is located near the bladder it may have to be manipu-

lated upward into a more accessible portion of the ureter. Removal of stones located in the portion of the ureter in the bladder wall is best accomplished by the transvesical route and the concrement extracted by splitting the ureter from within the bladder. When a stone is impacted in the vesical ureteral orifice it may, occasionally, be removed by means of the operating cystoscope; however, the method requires special skill.

The plastic operations on the ureter are described in connection with the surgery of contiguous organs (see also No. 30 of bibliography).

Diseases of the Suprarenal Glands.— Of the diseases of the suprarenal gland, eighty per cent are tubercuious in nature; the others consist in traumatic hemorrhages (Langmead[80]) and tumors.

Tumors possess surgical importance in the sense that they are exposed by the surgeon in connection with operations on the kidney. The influence that the removal of these lesions may have upon *Addison's disease* is indicated by recent experimentation, which seems to show that this mysterious affliction is not infrequently the result of involvement of the sympathetic ganglia and not the outcome of adrenal disease itself. See bibliography of Kümmel, No. 1.

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CHAPTER II

THE SURGERY OF THE BLADDER

Disturbances of the Bladder.— The clinical picture produced by disturbances of the bladder is an exceedingly varied one and is indicative of a variety of conditions. This is ascribable to the fact that lesions of the kidney produce symptoms referable to the bladder; that diseases of the prostate and of the posterior urethra are attended with similar disturbances; and, lastly, that diseases of the nervous system provoke manifestations referred to the bladder when the latter does not show the least evidence of anatomical changes. Diseases of the bladder are preëminently attended with disturbances of micturition.

Abnormal frequency of micturition (pollakiuria) is a symptom of importance only when it displays a certain persistence and extends over both day and night. In a number of instances this frequency, especially when due to stone or foreign body, is increased during locomotion and lessens when the patient is in repose. As frequency in micturition increases, in connection with a local lesion, the act of micturition becomes *precipitate* in the sense that the bladder must be emptied at once.

Pain in the bladder, other than that attendant upon micturition, is rare; it does, however, occur in connection with acute infectious cystitis. The pain attendant upon micturition may occur at the beginning of the act, at its end, or during its entirety.

Difficulty in micturition (dysuria) may be due to *mechanical obstruction* or to disturbances of innervation. The first lies in the neck of the bladder (prostatic hypertrophy), in the course of the urethra (stricture), or it may be due to calculi in the bladder. Dysuria is rarely a single symptom; usually it is associated with frequency of micturition.

The *urinary stream* is likely to be altered in caliber and projection when the vesical lesion is a mechanical one, such as a movable tumor or stone or when the expulsive activity of the organ is interfered with

in connection with nervous disorders. Nocturnal dysuria indicates prostatic disease; that of nervous origin persists both day and night.

Urinary retention may be complete, or may consist in inability entirely to empty the bladder; the retained urine is designated as *residual.* The retention may also be due to local or nervous causes. The former consist in prostatic hypertrophy or other lesion at the neck of the bladder; in women the altered relationship of the bladder to its outlet (cystocele) must be taken into account. In acute retention, the condition is self evident; in the chronic form micturition is apparently normal, but the urine voided is actually an *overflow;* in these instances the residual urine may be great in quantity (1000 g.).

Urinary incontinence is a term applied to involuntary discharge of urine. It occurs in connection with overflow (false incontinence) or, as is frequently the case, is a symptom of a lesion of the spinal cord.

Methods of Examination.— Inspection and percussion give little information with regard to the condition of the bladder; on the other hand, *palpation* is of distinct value in this connection. The distended bladder is readily felt, a sharply defined globular tumor above the symphysis. In the same way tumors of the bladder may be outlined and the increased resistance characteristic of prevesical infiltration (extraperitoneal rupture of the bladder, hematuria, paracystitis) is recognized.

Rectal examination permits palpation of the small portion of the bladder between the ductus deferentes and the rectum. It establishes the condition of the posterior wall of the bladder and gives valuable information with regard to the size and condition of the prostate gland.

Sounding of the bladder is accomplished by *elastic and by rigid* instruments. The elastic bougie is a thin straight instrument (silk) and has its end fashioned in the form of an olive. It may be introduced into the bladder without causing pain and conveys to the hand the varying sense of resistance imparted by pathological changes in the urethra and bladder and by the presence of calculi and foreign bodies; in the last, contact with a calculus is announced by an unmistakable sensation of friction.

The fallacy of an elastic instrument lies in the fact that it does not come in contact with the entire surface of the bladder and therefore the findings of the measure are not positive in a negative sense.

The *metal sound* (Fig. 1004) used for exploring the bladder has a curved beak like a catheter, and a hollow handle; the latter increases the resonance when the beak impinges upon a hard substance.

After the instrument is introduced into the bladder it is held in the horizontal position and its beak may then be turned on its axis to the right or left. The examination gives information with regard to the configuration of the bladder, the condition of its walls, and the presence of stone or of a foreign body.

Examination with the *catheter and syringe* gives valuable information as to the function of the bladder. The manner in which urine flows from the catheter indicates the functional activity of the detrusor, it reveals the amount and character of residual urine, and the sufficiency of the vesical musculature. Demonstration of the presence of residual urine is illuminating with respect to hypertrophy of the prostate or stricture of the urethra, and is of importance in connection with lesions of the nervous system.

In addition to this, the examination with the catheter decides the question of rupture of the bladder, especially when coagulated blood is withdrawn.

Injections of fluid into the bladder determines its capacity, while lavage of the bladder is helpful in recognizing the source of *hematuria*

Fig. 1004.— Bladder Searcher.

or of *pus*. The retained catheter assists in estimating the excretory capacity of the kidneys.

The *cystoscope*, which permits of visualizing the interior of the bladder, is by far the most valuable diagnostic instrument at the disposition of the surgeon. The instrument does not differ widely, in its construction, from the ureterocystoscope already described (p. 2270). As the full diameter of the instrument is available for the optical apparatus, it gives a wider field of vision than the ureterocystoscope; therein lies its essential difference from the latter.

In *cystoscopic examination* the instrument is introduced into the bladder under the same conditions as those spoken of in connection with ureterocystoscopy. In the effort to visualize bladder lesions, the surgeon is much handicapped by the persistence with which the field is obscured by blood and exudate from the bladder wall. As this often demands frequent lavage of the bladder during the examination, it is perhaps best, under these circumstances, to employ an irrigating cystoscope. Great care must be taken to avoid burning the bladder

wall. The introduction into the bladder of 0.5 per cent novocain solution makes the procedure bearable; in cases of great vesical irri-

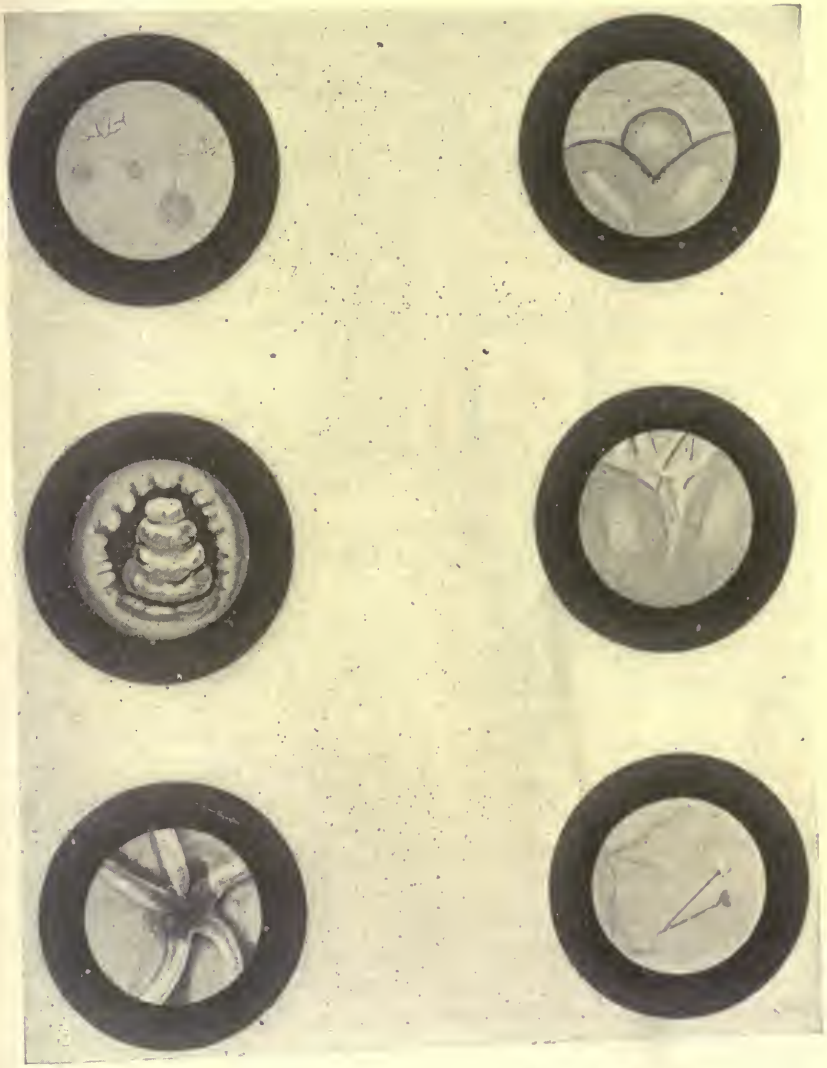

FIG. 1005.— CYSTOSCOPIC PICTURES (Keyes).

1, Early stage of tuberculous cystitis; 2, enlarged median lobe of prostate; 3, four encysted stones; 4, bilateral prostatic hypertrophy; 5, trabeculated bladder; diverticulum of lateral wall; 6, pin impaled in dome of bladder wall.

tability (ulcerative cystitis, tuberculosis, etc.) narcosis is indispensable. While the method is largely in the hands of the specialist, there is no reason why its technic should not be mastered by the surgeon. The details in this connection cannot, for obvious reasons, be presented here. The reader may gather an adequate notion of the character of the cystoscopic field presented under certain conditions by consulting the illustration shown in Fig. 1005.

Aside from the bladder conditions revealed by the cystoscope, *visualizing the ureteral openings* often permits of a conclusion as to the condition of the kidneys. In pyuria and hematuria the observer may see pus or blood, or both, issuing from the ureteral opening. Renal and ureteral tuberculosis is attended with characteristic changes in the mucosa contiguous to the ureteral opening. A stone impacted in the latter may be directly visualized, or its presence may be revealed by the characteristic deformation of the papilla.

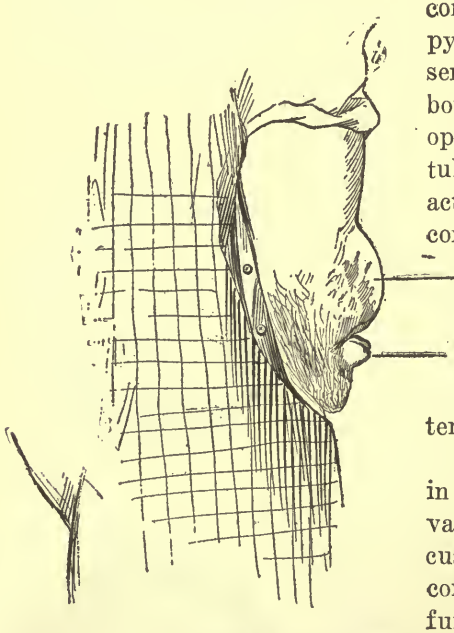

FIG. 1006.— EXSTROPHY OF THE BLADDER.

The *Röntgenographic findings* in lesions of the bladder are of less value than are the methods discussed above; there are, however, conditions in which the measure furnishes valuable information. Alterations in the position and form of the bladder (vesical hernia, diverticula, double bladder) may be reproduced on the negative by filling the organ with collargol. In cases of stone or foreign body the bladder should be filled with air. Recently, Zuckerkandl[1] has succeeded in obtaining the shadow of the prostate by filling the bladder with collargol and air (see also Stein and Stewart[2]).

Diagnostic suprabubic cystotomy is permissible when all other means to accomplish the purpose have failed. The measure is rarely necessary.

Congenital Malformations of the Bladder.— Vesical clefts are deform-

ities in which the anterior wall of the bladder is absent, so that the organ forms a flat surface, the mucosa of which is exposed. They are combined with clefts of the bony pelvis and the external genitals. The deformity is called *exstrophy of the bladder.* The open bladder lies immediately above the symphysis; the mucosa is wrinkled and thickened and bleeds easily. Two small papillae at the end of the defect correspond to the ureteral openings; these intermittently expel streams of urine. As a rule, the abdominal wall is defective in the region of the exstrophy and the deformity is always associated with arrest of development of the penis, scrotum, malposition of the testicles, etc. The bony pelvis as a whole is deformed and shows a defect at its anterior aspect. The skin contiguous to the extruded bladder is in a state of eczema, the outcome of the irritating effect of the urine with which it is constantly bathed.

Exstrophy of the bladder is the result of failure of coalescence of the abdominal wall in the process of development of the fetus. The variations in this regard and the embryological considerations are presented by Enderlen.[3]

The *symptoms* of the malformation relate to the irritation the exposed muscosa is subjected to by contact with clothing, etc., and the development of infection, which soon ascends in the course of the ureters and is responsible for pyonephritis; the latter is usually followed by a fatal outcome. On the other hand, not a few cases are on record in which the afflicted person has carried his burden well along into adult life.

The problem in the *treatment* of exstrophy of the bladder consists in the conversion of a small sector of vesical mucosa into a hollow organ which permits of the accumulation of urine and its intermittent expulsion. While surgery has accomplished certain triumphs in this connection, none of these has been entirely satisfactory.

The efforts at repair have consisted in the *operative closure of the defect* with pedunculated skin flaps taken from the areas contiguous to the malformation. This is, in some instances, preceded by a free dissection of the mucosa and the approximation of its refreshed edges (see v. Eiselsberg[4]). Trendelenburg[5] employed a series of complicated procedures destined to produce conditions which approximated the normal. The outcome made the affliction less offensive.

A comparatively new thought is presented by efforts to *lead the flow of urine into other channels* and thus eliminate the distressing disturbances consequent to the constant discharge of urine. These efforts

found their culmination in the procedure practiced by Maydl,[6] who mobilized the malformed bladder, resected it, except for the trigone, and implanted the latter into the sigmoid. The important factor in the procedure is that it preserves the oblique entrance of the ureters with their sphincteric action, and prevents ascending infection of the urinary passages from the gut. The various minor modifications of the method are summarized by v. Eiselsberg.[4]

Absence of the bladder is associated with other malformations of the pelvis. The ureters usually open into the urethra.

Fetal dilatation of the bladder occurs in connection with obstructive anomalies of the urethra.

Double bladder is a term applied to a two chambered organ with a ureter opening into each of the sectors, which in turn open separately into the urethra. More commonly, the bladder sectors communicate with one another and only one opens into the urethra (pocketed bladder).

The *diagnosis* is readily made with the cystoscope and by Röntgenography of the bladder injected with collargol.

Resection of an infected sector and implantation of its ureter into the remaining one has been several times successfully practiced (Zuckerkandl[1]).

Injuries of the Bladder.— Vesical injuries are divided into *rupture* and *wounds;* the former is produced by bursting of the bladder, the outcome of increased intravesical pressure. Wounds of the bladder are classified as subcutaneous and penetrating.

Rupture of the bladder is divided into *traumatic* and the so-called *spontaneous;* the latter is caused or predisposed to by a pathological process.

The bladder wall bursts when the intravesical pressure exceeds the limit of the distensibility of the organ; the increase of pressure may occur in connection with extrinsic influences. As a rule, a full bladder is subjected to a sudden force, such as a kick, a fall, the impact of a heavy body, the blow of a falling weight, the compression of a wagon wheel, crushing between buffers, etc., and in most instances a simultaneous fracture of the bony pelvis occurs. Occasionally, the bladder is ruptured by the compression of the abdominal muscles during great muscular exertion (lifting, tumbling, etc.). While it is possible that the bladder may be ruptured from overdistention with urine, this is highly improbable. Occasionally, a pathological bladder, especially

one with a diverticulum, is ruptured in connection with forcible distention employed in extracting *detritus* after *lithotripsy*.

As already stated, spontaneous rupture of the bladder may occur in connection with pathological changes in the vesical wall, such as tuberculosis, carcinoma, diverticula, etc. It also occurs when the bladder is paralyzed in cases of lesions of the spinal cord.

All forms of *rupture* of bladder occur *when the organ is filled* with urine. The empty bladder is rarely injured, although this may occur in gunshot and stab wounds and in so-called impalement injuries (p. 2170).

Ruptures of the bladder are linear or stellate in form. They are divided into *intraperitoneal* and *extraperitoneal*, or these may be combined.

Intraperitoneal rupture is usually located at the posterior wall of the organ and is the most frequent form of the lesion. In this form the urine enters the abdominal cavity and collects in the pelvis. When the urine is infectious, peritonitis promptly develops; the latter appears late when the urine is sterile. The peritoneal inflammation may be localized or diffuse.

Extraperitoneal rupture usually occurs in the anterior wall of the bladder. The urine enters the paravesical cellular tissue and collects there in the form of a globular tumor; ultimately, a paravesical phlegmon is formed. Bartels[7] has collected and analyzed 16 cases of rupture of the bladder.

The *symptoms of rupture* of the bladder are constitutional and local. The former are those of shock, the degree of which varies with the severity of the injury.

The *local symptoms* consist in pain in the lower abdomen and frequent and futile efforts at urination. What little urine is voided is tinged with blood, or only a few drops of blood are expelled.

The region above the symphysis is tender. Catheterization usually withdraws only blood. On the other hand, if the catheter should happen to pass through the rent in the bladder a large quantity of bloody or clear urine may suddenly be expelled.

In *intraperitoneal rupture* the symptoms of peritonitis soon develop. This is usually fatal.

In *extraperitoneal rupture* the subsequent course is that of gradual urinary infiltration and phlegmonous inflammation.

The *diagnosis* in clearly defined cases is self evident. In some instances it is difficult to differentiate vesical from renal injury, espe-

cially when hemorrhage and reflex anuria occur together. Rupture of the urethra is attended with retention of urine, which is readily made out. Examination with the catheter is the most helpful measure; withdrawal of a small quantity of bloody urine after prolonged retention is almost pathognomonic of rupture of the bladder. This is the only permissible intravesical manipulation; *lavage of the bladder* is attended with the danger of infecting the peritoneum and is *contra-indicated* (Horwitz[8]).

The *treatment* of rupture of the bladder consists in exposure of the lesion, removal of the extravasated urine, repair of the lesion and the placing of the organ out of function until healing is accomplished. The first successful surgical repair of the bladder was made by Walter[9] of Pittsburg in 1878.

In all cases of (intra- or extraperitoneal) rupture of the bladder suprapubic exposure is indicated. The rent in the bladder is closed with catgut in layers as in intestinal suture. A retention catheter is kept *in situ* for five or six days (see suprapubic cystotomy).

Wounds of the bladder do not present problems other than those taken up in connection with rupture. In gunshot wounds contiguous viscera are very likely to be simultaneously injured, for obvious reasons. In wounds of this nature and in those produced by instruments of various kinds, a portion of the object may be retained in the bladder and form the nucleus of a stone.

Primary healing of wounds of the bladder is rare. The infection attendant upon extravasation of urine is usually responsible for more or less inflammation and this leads to the formation of *fistulae*. Many of these heal spontaneously; others demand plastic measures of repair.

Foreign Bodies in the Bladder.— Foreign bodies reach the bladder through the urethra, through a wound, or enter it by ulceration from a contiguous organ. Those introduced through the urethra are most often surgical instruments used in this location, which accidentally slip into the bladder (portion of a catheter, filiform guides, fragments of a lithotrite, etc.). A considerable number of bodies introduced in this way gain access to the bladder during erotic manipulations; these are usually hairpins, penholders, lead pencils or other round objects. As already stated, projectiles, portions of clothing, or pointed metal blades, etc., may enter the bladder through a wound. Silk ligatures used during pelvic operations and uterine pessaries, when worn for long time, not infrequently ulcerate into the bladder.

Foreign bodies, as such, do not give rise to disturbances; they are,

however, carriers of infection and thus often stand in a causative relationship to severe inflammatory processes in the bladder. Pointed bodies may become embedded in the bladder wall and may be responsible for infection of the paravesical tissues.

Small bodies may be expelled with the urinary stream. An impacted body may perforate the bladder wall and in this way gain access to the gut or vagina. When the peritoneum is thus invaded fatal peritonitis may ensue.

Fig. 1007.—Phosphatic Calculus formed upon a Twig (Keyes).

Foreign bodies retained in long contact with the urine undergo certain changes; vegetable substances disintegrate; resisting ones are gradually incrusted with the salts contained in the urine. Glass does not become incrusted; rubber, iron, and vegetable substances are rapidly covered. The phosphates either simply incrust the body or completely obliterate its form, so that it simulates a calculus (Fig. 1007).

The *symptoms* of foreign bodies in the bladder are those consequent to mechanical interference with micturition, and to the inflammatory processes their presence is responsible for. Frequent and painful micturition, retention, dribbling and pain in the intervals between micturition are characteristic symptoms.

The *diagnosis* is frequently made on the history of the case. In obscure cases the cystoscope determines the question. The searcher may reveal the presence of the body. The Röntgenographic findings, unverified by the cystoscope, are very misleading.

The *treatment* consists in removal of the unwelcome visitor. Efforts at dissolving the body and its removal by lavage are not often successful. The ideal treatment is to remove the body through the urethra;

this is occasionally feasible by means of especially constructed instruments fashioned not unlike a lithotrite. The modern method of accomplishing the purpose with the operating cystoscope is very likely to succeed when the body is not too large. Failing in this, the offending substance is removed through a suprapubic cystotomy.

Diseases of the Bladder — Cystitis.— The *causation of cystitis* presents a manifold problem. Inflammatory processes of the bladder are the outcome of the action of pathogenic organisms or their products. However, the presence of microörganisms in the urine may not produce inflammation of the bladder. The urine may contain pure cultures of bacteria (*bacteriuria*) and the bladder remain uninfected. Bacterial reaction presupposes a certain predisposition on the part of the bladder in this connection; these are traumatic epithelial defects, prolonged congestion, modifications in the niveau of the interior of the bladder, trabeculous hypertrophies, diverticula, a prominent body of the uterus, and all the various forms of urinary retention. Experimentally, the normal mucosa of the bladder possesses a certain immunity against bacterial infection.

In children, the *congenital* forms of urinary obstruction, infection with the *communis coli,* and the *acute infectious diseases* are often responsible for cystitis.

In the adult, *gonorrhea* and *tuberculosis* and, late in life, *prostatic hypertrophy* are the most common causes of the disease.

Bacteria gain access to the bladder in various ways. The bacteria filtered from the blood by the kidney (typhoid and tubercle bacilli, etc.) enter the bladder with the urine and, in some instances, the organ is infected secondarily to pyogenous diseases of the kidney. However, as already stated, the bladder is not infected unless it is particularly receptive in this connection. This type of infection is called *descending.*

Infection from the urethra (*ascending*) occurs in connection with suppurative prostatitis, unsterile instrumentation, and lavage.

Contiguous suppurative foci (in the Pouch of Douglas, adnexal infection, paracolitis, etc.) may occur without a solution of continuity of the bladder wall (bacterial transmigration).

Hematogenous cystitis, such as develops in miliary tuberculosis, is possible but possesses little clinical significance.

Anatomically, cystitis is classified as (a) superficial, (b) interstitial, (c) perivesical.

The *superficial* is the least severe and usually involves the neck,

trigone, and the mucosa in the vicinity of the ureteral orifices. If continued, this variety of the disease merges into the *interstitial* type, involving the outer coats of the bladder in addition to the mucous membrane with resultant swelling and thickening of the affected parts and contraction of the bladder. The mucosa in both instances becomes reddened, edematous, and often ecchymotic. At times the membrane becomes rough in appearance, producing a prominence of the folds of the mucosa (trabeculated bladder).

Clinically, cystitis is divided into *acute* and *chronic* forms; *pathologically* these are divided into (a) catarrhal, (b) suppurative, (c) ulcerative, (d) exudative, and (e) exfoliative (Senn[10]).

In *catarrhal cystitis* the process is superficial and the urine contains a predominance of epithelial cells rather than pus. In *suppurative cystitis*, in addition to the superficial inflammation, the deeper interstitial tissue is involved; this is attended with excessive secretion of pus, the destruction of epithelial cells, and the deposit of fibrinous exudate in areas which display a tendency to necrose. The urine is strongly ammoniacal.

In *ulcerative cystitis* the lesion is destructive at the onset. The ulcers are regarded as due to a hematogenous infection, i. e., septic embolus lodges in the bladder wall and the ulcer extends by destructive inflammation.

Exudative cystitis is a form of coagulation necrosis. It occurs most frequently in puerperal women and is characterized by the presence in the urine of membranous shreds of fibrin.

Exfoliative cystitis is ascribed to the action of toxins. The vesical membrane is destroyed and thrown off in the form of a mold of the bladder (Stein[11]).

The *symptoms* of cystitis may be stated as consisting of pyuria, frequency and urgency of micturition, hematuria and pain. As has been frequently stated above, none of these is diagnostic in a sense of localization. The urine is turbid and, when allowed to stand, shows a sediment consisting of mucus and mucopus mixed with epithelial cysts, crystals, and a number of bacteria; the reaction is usually alkaline. In colon infection the urine is acid. In acute cystitis, pain is a prominent feature; it is referred to the perineum and the hypogastrium, and is accompanied by great vesical irritability, often exacerbated to strangury. A fatal outcome, due to the virulence of the infection, exhaustion, peritonitis or acute suppurative pyonephritis, is not uncommon.

The *diagnosis* is certain only when established by cystoscopy.

The *prognosis* in acute cystitis is not unfavorable; it is, of course, dependent upon the causative factor. In the chronic form of the disease an unfavorable outcome is likely to be caused by secondary changes in the kidney, etc.

Considering the large number of instances in which cystitis is due to instrumentation as compared to the so-called spontaneous cases, the first duty in the *treatment* of the affliction relates to the exercise of asepsis in this connection. When all urological instruments are sterile the number of cases of cystitis is much reduced.

The *treatment of acute cystitis* is directed toward shortening the course of the disease. The patient is put to bed, hot applications are made to the bladder and a bland diet, together with large quantities of water, are administered. The ingestion of urinary antiseptics (salicin, benzoin, camphor, and salol) is largely employed on the theory that they are eliminated in the urine; their prolonged use may give rise to carbolic intoxication. On the other hand, formaldehyd splitting agents are more effective and do not cause objectionable disturbances; of these urotropin and its derivatives still occupy first place. The local treatment of acute cystitis is not well borne. Catheterization is permissible only for the relief of retention of urine.

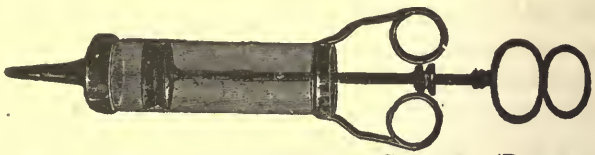

FIG. 1008.—A LARGE PISTON SYRINGE (BLADDER SYRINGE) USED FOR URETHROVESICAL IRRIGATIONS AND WASHING OUT THE BLADDER THROUGH A CATHETER.

In *chronic cystitis* the local treatment dominates the situation. The principle of the measure lies in the artificial evacuation and lavage of the bladder.

The *bladder is emptied* by means of a soft rubber catheter, when the residual urine is of considerable quantity, on the ground that the stagnation of infected urine is the chief factor in the persistence of the disease. The bladder is evacuated as often as the symptoms demand, i. e., to accomplish recession of tenesmus and frequency of urination. If fever persists, permanent drainage with the retention catheter is indicated. In severe, prolonged cases the establishment of a suprapubic fistula is permissible. In other words, the principle of providing ready egress for infective exudate is to be applied in this connection in the same way as in other portions of the body.

Lavage of the bladder aims at removal of purulent secretion stagnated in the niches and adherent to the loose wall of the organ. The measure acts mechanically; the use of nonirritating antiseptic solutions is not objectionable.

The fluid is permitted to enter the bladder by gravity from an irrigating container, which it is not necessary to depict here, or a syringe

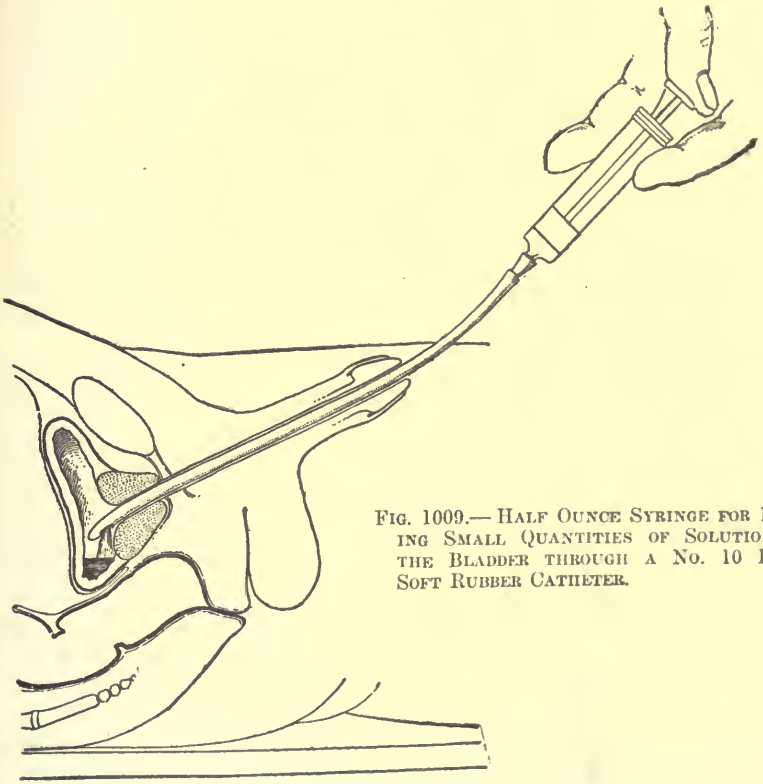

FIG. 1009.— HALF OUNCE SYRINGE FOR INJECTING SMALL QUANTITIES OF SOLUTION INTO THE BLADDER THROUGH A NO. 10 FRENCH SOFT RUBBER CATHETER.

may be used to inject the fluid (Fig. 1008). The solutions most frequently employed for the purpose are 3 per cent boric acid; hydrargyrum oxycyanidum 1:4000; and argentum nitricum 1:4000. Preliminary injection of novocain (0.5 per cent) makes the measure painless.

The *drop instillation* of medicinal agents is indicated when lavage is not tolerated. The type of instrument used for the purpose is shown in figure 1009. The olive pointed hollow tube is gently introduced into

the bladder and small amounts (15 cc.) of silver nitrate, as strong as 2 per cent, may be instilled into the inflamed organ.

The *retention catheter* is the sovereign measure of relief in chronic cystitis and urinary retention of high degree. It need not be continuously employed; for instance, it may be introduced only at night. The measure must be carried out under strict asepsis. The catheter is connected with a suitable receptacle fastened to the bed.

Tuberculosis of the Bladder.— Tuberculous cystitis is probably never primary; it occurs either in connection with the general miliary form of the disease or the organ is infected from the kidney (see tuberculosis of the kidney, p. 2304). Tuberculous infection of the bladder from the male genitals does not seem to occur.

The first changes in the bladder consequent to renal tuberculosis take place in the mucosa at the ureteral papilla on the diseased side. The ureteral ostium loses its slit-like or punctate form, and swells; the contiguous mucosa is reddened and swollen, while the ostium becomes rigid and gaping. Soon a number of miliary tubercles fleck the neighboring mucosa; these finally coalesce and form a single, irregular, cheezy ulcer. The ulcerative process is for some time restricted to the side of the diseased kidney, but ultimately spreads, and finally the entire bladder presents the picture common to tuberculosis of mucous membranes (p. 431). As the process progresses, the bladder as a whole becomes the seat of chronic inflammatory thickening, which is not infrequently attended with shrinkage of the organ. In some instances the ulcerative process persists and extends over a greater or less area of the bladder wall.

Of the *symptoms* of vesical tuberculosis, Lewis[12] says "While it has not yet been definitely proved, it is my own belief that one of the earliest prodromes of urinary tuberculosis is the persistent appearance in the urine of red blood cells in microscopic quantity, covering a period of years, perhaps, before the disease reaches a stage which introduces its clinical features or changes to a macroscopic hematuria."

Undue frequency of micturition is one of the most important and characteristic symptoms, and may be observed before the secondary infection clouds the urine and before marked manifestations of tuberculosis are observable. It is produced by irritation, by direct involvement, or is reflex. When the lesion is confined to the vault of the bladder, these symptoms occur late. Pain in the bladder and neighboring regions is another feature of vesical tuberculosis, especially in the later stages. The pain is aggravated by urination and is referred to

the neck of the bladder, the perineum, the urethra, and the glans penis. The urine may be clear until secondary infection occurs and a most careful examination of the centrifugalized specimen must be made to explain the symptoms. Later, secondary infection causes the urine to become clouded and purulent, with shreds and strings of mucopus and particles of blood clots, and at times it assumes a bloody appearance, either intermittently or persistently. The general condition varies with the progress of the disease.

The *diagnosis* must be based on the discovery of the tubercle bacilli, together with direct observation of the process by means of the cystoscope.

In the *treatment* the general measures are the same as those employed in the care of tuberculosis elsewhere in the body. *Locally,* Lewis[12] uses iodoform emulsion introduced together with air through the cystoscope. Garceau[13] applies bichlorid of mercury and silver nitrate directly to the ulcerative area, with the aid of the cystoscope; general narcosis is required. Unilateral nephrectomy for tuberculosis of the kidney has beneficial results; when both kidneys are involved, removal of the one most diseased is followed by improvement of the vesical condition.

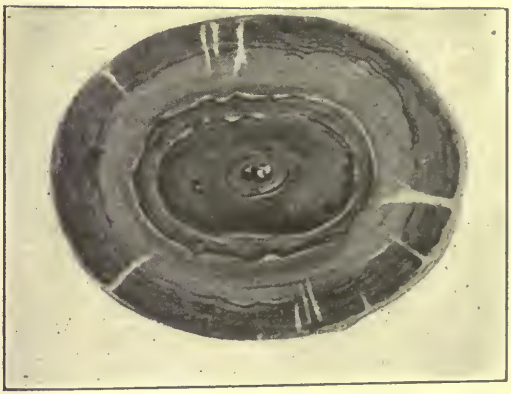

FIG. 1010.— URIC ACID VESICAL CALCULUS.

When pain is severe the establishment of a suprapubic fistula is permissible.

CALCULI OF THE BLADDER.— The *causation of vesical calculi* has been taken up in connection with nephrolithiasis and foreign bodies in the bladder. In a general way, one may say that the formation of stone is predisposed to by any change in the *form of the bladder which modifies the symmetrical smoothness of its mucosa.* These are: Niches between bundles of muscle fibers; ditches formed by a prominent body of the uterus; enlarged prostate, etc. The influence of the organic

material thrown off in connection with inflammatory processes is important in a causative sense.

Vesical calculi are classified as follows:

A uric acid calculus (Fig. 1010) is usually an oval, flattened stone of considerable density, nut brown in color, and smooth, or with a slightly nodular surface. On cross section it is laminated and may be incrusted with phosphates.

The *oxalate of lime or mulberry calculus* (Fig. 1011) has a rough, irregular surface, but may be symmetrically nodular; it is less frequently trabeculated. This form of stone is hard, laminated and dark red or brown in color; it is black if mixed with blood.

FIG. 1011.— OXALATE OF LIME OR MULBERRY VESICAL CALCULUS.

A *pure phosphate calculus* (Fig. 1012) is very uncommon. A foreign body in the bladder is certain to be coated with a phosphatic deposit when chronic cystitis results in alkaline decomposition of the urine. This type of calculus forms spontaneously in saccules of the bladder; it is chalky and friable with little or no lamination.

In rare instances, *cystine and xanthine* form the basis of calculi; the former is yellow green and the latter reddish in color.

A *vesical calculus* consists in a *nucleus,* which may be formed by a blood clot, foreign body, renal calculus, inspissated mucus; a *body,* which consists of superimposed layers of the substances determining its character; and a *crust,* which varies in amount and consists of a

friable phosphatic material — this is not always present. The number of calculi in a given bladder varies greatly. As a rule, multiple calculi are faceted.

An *encysted calculus* is one which develops in a pouch or pocket of the bladder wall.

The *symptoms* produced by a vesical calculus depend upon its size and shape and the tolerance of the mucous membrane. A history of renal colic is usually obtainable, if the nucleus of the stone is renal in origin. A stone which forms in the bladder may reach a large size before it gives rise to disturbances, while small ones may cause enough irritation to give rise to aggravated symptoms. Usually, *increased frequency* of urination occurs early in cases of stone and may become so marked that the patient urinates every few minutes. *Sudden stoppage* of the flow of urine occurs when the stone comes in contact with the internal orifice of the urethra during urination. This is accompanied by a sharp pain and occurs most frequently in youth and middle age, when the urethral orifice has not been raised by enlargement of the prostate. *Pain* varies in intensity, but has certain characteristics which make it a valuable aid in diagnosis. It may occur as a general sense of weight and discomfort above the pubes, in the perineum or the rectum. When it becomes more severe, the pain is cutting or pricking, and shows a tendency to radiate along the urethra; or it is referred to the end of the penis. Pain is aggravated at the end of urination and by motion, such as being jolted in a vehicle; this also occurs in walking or even while turning in bed. Reflex pains are referred to the hip and thigh, and, at times, to the lower leg and foot.

FIG. 1012.— PHOSPHATE OF LIME CALCULUS.

The *urine*, early in the process when vesical irritation is not marked, may be clear to the naked eye. However, microscopical examination reveals the presence of blood and pus, together with crystals of uric acid and oxalate of lime in varying amounts. As the process progresses the urine becomes more and more turbid, and, in some instances,

is colored by the admixture of blood. When the urine becomes alkaline its pus content becomes gelatinous and at times is tinged with blood. Hematuria may be severe enough to be attended with clotting. Occasionally blood appears only at the end of urination and may be large in quantity.

Fig. 1013.—Bigelow's Lithotrite Opened.

The *diagnosis* is not always easy. Differentiation is based on the physical examination.

(a) *Digital examination* (bimanual) via the rectum or the vagina may reveal the presence of a stone. (b) Exploration with the *searcher* and *lithotrite* makes the diagnosis — the metal beak coming in contact with the stone gives rise to a "click." A negative finding does not exclude stone. (c) For small stones which evade the searcher, the litholapaxy pump of Bigelow may be used to wash out the calculi. (d) The cystoscope is positive in its results. (e) The Röntgenogram may reveal the presence of an encysted stone. (f) Exploratory incision (suprapubic) is permissible when other means are not available.

The *surgical treatment* of stone in the bladder consists in its removal.

Lithotrity in one sitting owes its origin to Bigelow[14] (1878). Preliminary control of the complicating cystitis is desirable. After the patient is narcotized the bladder is lavaged until the return is clear. About six ounces of the fluid is left *in situ* and the lithotrite (Fig. 1013) is introduced. In inserting the instrument the handle must be well depressed between the thighs in order to pass the beak under the pubic arch. The position of the stone is ascertained by rotation of the instrument, using it as a sound. The blades are opened and the stones caused to roll between them by a slight jerk of the handle, which is a better and less dangerous method than attempting to invert the blades and pick up the stone. If the stone is fairly grasped as the blades are screwed together, it is crushed into several fragments, each of which is treated as is the original stone. When the operator is reasonably certain that the fragments are small enough, the largest tube that can be passed with safety is introduced into the bladder.

The evacuator is attached to the tube and the bladder thoroughly washed out by alternating pressure upon and relaxation of the rubber bulb. The fragments of stone are collected in the glass receptacle below the rubber bulb (Fig. 1014). The washing is continued until no more fragments are heard or felt to rattle against the end of the tube. At times, some of the remaining fragments are too large and the lithotrite must be reintroduced and the crushing process

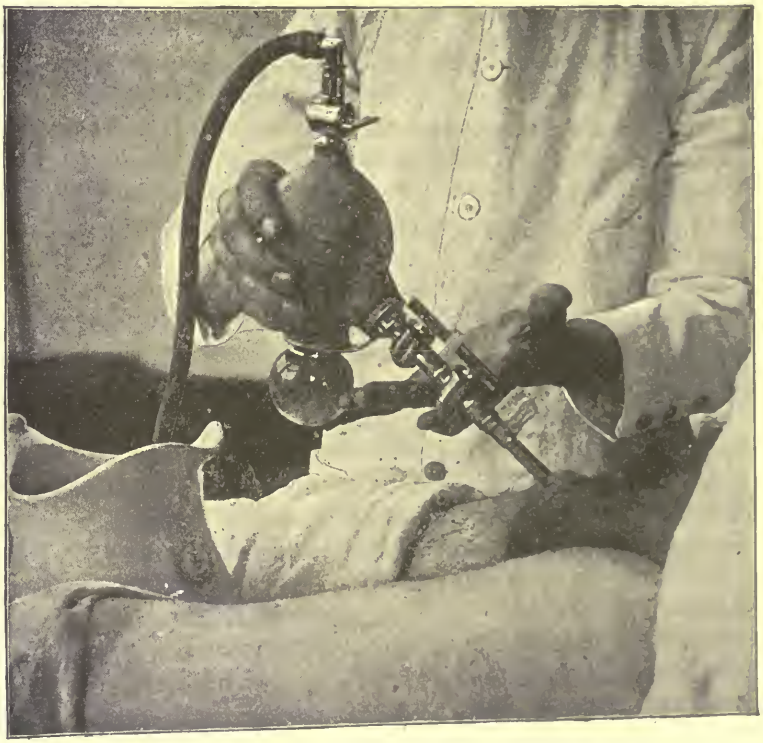

Fig. 1014.— Bigelow's Apparatus in Action.

repeated. The maneuver is attended with some hemorrhage, but with careful manipulation it is not excessive. The patient is kept in bed for one week.

Removal of a stone by *suprapubic lithotomy* is executed as follows: With the patient in the Trendelenburg position, a vertical incision is made just above the pubes and the bladder is exposed. The latter is held in place by two retention sutures and incised. The finger is intro-

duced into the bladder and the stone is removed. The bladder is drained through the suprapubic wound, which heals by granulation in from three to six weeks.

Perineal cystotomy is rarely employed for the purpose of removing a calculus. The procedure does not differ from that employed in connection with external urethrotomy. The various lateral perineal incisions possess only historical interest. See bibliography of Zuckerkandl.[1]

Lithrotity is the measure of choice, the contraindications being (1) a calculus exceeding one and one half inches in diameter; a hard stone consisting of oxalate of lime; an encysted stone; (2) organic stricture of the urethra or an enlarged prostate; (3) severe cystitis, the presence of sacculi; a markedly contracted bladder.

Suprabubic lithotomy is contraindicated only by coincident severe septic cystitis and contraction of the bladder. It is indicated in the presence of the contraindications for lithotrity.

Perineal lithotomy is indicated in the event of severe cystitis and great vesical irritability (the method furnishes excellent drainage and facilities for irrigation), and in cases of contracted and hypertrophied bladder, or of impacted calculus in the neck of the bladder.

Calculus in the female (if one half to three quarters of an inch in diameter) may be extracted through the dilated urethra with the finger, nicking the sphincter in two or three places. If the stone is larger it is removed via the suprapubic route. *Vaginal lithotomy* is objectionable on the ground that a vesicovaginal fistula often follows the measure.

Tumor of the Bladder.— In the *etiology of new growths* of the bladder protracted irritation plays an unmistakable rôle. The epithelium of the bladder, like that of the entire urinary tract, responds to irritation in the form of proliferation and changes in the character of the cells to a greater degree than that of other organs of the body. It is readily converted into pavement epithelium and in response to slight irritation forms cysts or becomes the seat of glandular multiplication.

This tendency on the part of the vesical epithelium, toward multiple metaplasia, explains the frequency of epithelial tumors, while the number of cases of sarcoma of the bladder is very small. The fact that the majority of vesical tumors develop at the base of the bladder, and that small papillary growths are so often found near the ureteral openings, suggests that the muscular activity of the organ in this situation contributes a certain traumatic element in the causation of this

class of lesions. However, the most important predisposing factor in this connection lies in the irritation coincident to *chronic inflammatory processes.* The transformation of cystitis cystica and glandularis into mucous or glandular carcinoma is an established histological fact. The influence of intensive chronic cystitis upon the development of new growths is best studied in the ectopic bladder, especially as glandular carcinomata most often originate in the exposed organ, in which inflammatory processes are attended with the active formation of glandular tubules.

The etiological significance of bilharziasis of the bladder, in connection with new growths, is firmly established. The chronic inflammation produced by this fluke worm is undoubtedly responsible for epithelial proliferation which, according to Grebel,[15] is carcinomatous in character. The causative influence of *chemical irritation* is shown by Rehn,[16] and that of stone complicated by chronic cystitis is quite accepted.

Papilloma (villous pedunculated) of the mucosa is the most frequent tumor found in the bladder. The tumors to which the term *papillomata* is applied do not present the same histological picture. The view of Rokitansky,[17] that these growths are all cancerous (villous carcinoma), has been much contested, yet, in the light of recent investigation, seems to possess considerable justification (v. Frisch[18]). This class of growth is discussed in Part VI, chap, xvi.

From a *clinical standpoint* it is important to realize that all forms of papillomata show a distinct tendency to become malignant. Zuckerkandl[1] presents an interesting analysis showing that MOST OF THE PAPILLOMATA OF THE BLADDER, WHICH GAVE NO CLINICAL EVIDENCE OF MALIGNANCY, PROVED, ON MICROSCOPICAL EXAMINATION, TO BE CARCINOMATA.

In *carcinoma* of the bladder the epithelium and stroma show irregular growths. Metastases in distant organs are common.

As already stated, carcinoma may make its appearance in the guise of papilloma; in other instances it develops in one of the forms discussed in connection with carcinomata of mucous membranes (Part VI, chap. xvii). In not a few cases the growth is flat; in others it is nodular or ulcerative.

As the growth extends, it invades the prostate, the vesiculae seminales, and infiltrates the paravesical tissues, so that the bladder is a rigid organ. As a rule, the process grows slowly and metastases occur late. Like papillomata, carcinomata usually originate at the base of

the bladder; occasionally they arise in the dome of the organ. In not a few instances the new growth obstructs the ureteral openings; this leads to dilatation of the ureters and kidney pelves.

Adenomata develop in the glandular tubules of the mucosa in much the same way as in the gut (Zuckerkandl[1]).

Mixed tumors of the bladder are rare. They are derived from the middle layer of the embryonic anlage and are connected with the wolffian duct.

Dermoids are also rare.

Connective tissue tumors (sarcomata, myomata, fibromata, myxomata) originate much less frequently in the bladder than do epithelial growths. Clinically *sarcomata* cannot be differentiated from carcinomata of the ulcerating type. They are usually primary.

Of the *symptoms of tumor of the bladder,* hematuria is the most frequent initial disturbance noted, irrespective of the character of the growths. The bleeding occurs without assignable cause and may be profuse, may continue for a long time and then suddenly cease, to recur in the same way. In the intervals the urine is likely to be perfectly clear. The bloody urine is usually discharged without distress. Examination of the urine does not disclose the presence of characteristic constituents, except in rare instances in which a fragment of the growth is voided. In ulcerating carcinoma the urine may emit an offensive odor. In papillomata the urine is aseptic; in ulcerating carcinoma it contains pus. Only tumors located near the urethral orifice provoke disturbances in micturition. Late in the process, when the ureteral openings are more or less occluded, kidney pain and colic make their appearance.

The *diagnosis* of vesical tumor is not indicated by a characteristic sign or symptom. Today, the occurrence of hematuria is regarded as justifying examination of the bladder with the cystoscope, which establishes the presence of a tumor, but does not, of course, determine its character. Papillomata may be identified by their characteristic appearance and by microscopical examination of a fragment removed by lavage. In doubtful cases, an effort may be made to remove a sector of the growth for examination, employing the operating cystoscope for the purpose.

In view of the doubtful character of vesical tumors, conservative *treatment* cannot be regarded as meeting the indications. On general principles all tumors of the bladder should be extirpated.

Extirpation of a vesical tumor by cystotomy is not a difficult pro-

cedure, provided the suprapubic incision is ample. The surgeon must not err on the side of timidity in this connection. The sector of the bladder to which the tumor is attached should be excised. If this happens to correspond to the ureteral openings, the maneuver may present considerable technical difficulty. Large papillomata may have to be removed by *morcellement;* the residual stump is excised, together with a goodly sector of mucosa. In many instances, the mucosa may be separated from the ureter without injury to the latter. If, in order to gain the necessary space, it becomes necessary to open the peritoneum, this may be done as in surgical attack upon any other organ covered by this membrane.

Total extirpation of the bladder is attended with great technical difficulties, but has frequently been accomplished with success. The rule is to transplant the ureters into the gut, or elsewhere, at one sitting, and extirpate the bladder several weeks later. See v. Frisch.[18]

Of the various situations into which the ureters may be implanted, attachment to the skin is least likely to be followed by ascending infection. The lumbar region is the location of choice.

The *endovesical extirpation of tumors of the bladder* is accomplished through the uretha by means of an especially devised cystoscopic armamentarium. The method is, perhaps, permissible in cases of pedunculated papillomata of small size. The endovesical treatment of papillomata with the *high frequency current* removes the growth by "heat coagulation" and destruction. The maneuver is very tedious, requires much time and not a little skill, and has a restricted field of usefulness.

The *palliative measure of relief* consists in the establishment of a vesical fistula; as it does not divert all of the urine, its accomplishment is only partial.

Vesical hernia is taken up in Part XVI, chap. xii.

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CHAPTER III

THE SURGERY OF THE PROSTATE

(GLANDULA PROSTATICA)

Method of Examination.— The prostate is *examined* in several ways. *Palpation* through the rectum (with the latter previously emptied) is best employed with the patient in the knee chest position. The measure is not painful. After passing the sphincter the finger glides over the bulb and membranous urethra; these are only indistinctly made out. The point of the prostate is next felt and beyond this the two lobes of the gland are readily recognized. In many instances the palpating finger determines the outlines of the seminal vesicles. When pressure upon the prostate causes its secretion to emanate from the urethra, this may be subjected to microscopical examination. The simultaneous introduction of a sound into the urethra gives information as to the location of the prostatic urethra and the internal orifice of the latter.

Especially constructed cystoscopes make it feasible to visualize the prostate. The *urethroscope* reveals the condition of the internal sphincter and the size and form of the prostatic urethra.

Congenital Malformations.— Congenital malformations of the prostate do not possess great surgical importance; they are usually a part of other developmental anomalies of the external genitals of high degree. The anomaly may consist in *total or partial absence of the gland.* Misplaced masses of the prostate, like those of the thyroid or mammary glands, have been observed by a number of surgeons (Young[1]). These explain the occurrence of pure glandular carcinoma in the wall of the bladder (Socin and Burkhardt[2]).

Prostatic Cysts.— Cysts of prostatic origin are not uncommon (English[3]). They are regarded as the outcome of obstruction of the urethral outlet of the sinus pocularis. They may be congenital or acquired; it is permissible to regard them as *retention cysts.* At times, they grow to sufficient size to obstruct the flow of urine. In this event they are very likely to be ruptured in the course of catheterization.

Another class of cysts develops in the *connective tissue between the prostate and the rectum or the bladder.* According to English,[3] these develop from embronic rests of Müller's duct or of the wolffian body or consist in dilatations of the vas deferens. These cysts often develop to the size of a pigeon's egg and are likely to give rise to disturbances in urination.

The *diagnosis* is not easy; especially as the cysts closely simulate diverticula. Their removal is accomplished through a suprapubic cystotomy wound or a perineal section, according to their location.

Injuries of the Prostate.— While, because of its protected position, the prostate is rarely injured, it is not infrequently torn from the urethra in fractures of the pelvis. Direct involvement of the gland occurs in connection with so-called *impalement injuries.* Gunshot wounds of the prostate are also very rare. On the other hand, the organ is not uncommonly injured during instrumentation. Bleeding is likely to be severe.

The injury is *treated* on general surgical principles. In all cases provision must be made for free discharge of the urine and wound secretions, employing external urethrotomy if necessary.

Inflammations of the Prostate.— Acute prostatitis is the outcome of bacterial infection. From this standpoint all other influences, such as cold, trauma, severe exertion, etc., become of minor importance, and probably are only capable of exacerbating a previously existing inflammatory process. This is also, in a measure, true of ungentle instrumentation, etc. No doubt inflammatory processes may extend to the prostate by way of the lymphatics from the rectum, periproctic tissues, bladder, and other pelvic organs. A certain number of cases are due to hematogenous infection.

By far the most frequent *cause of prostatitis* is *gonorrhea,* in both the acute and chronic form. As in gonorrheal rheumatism, the tissues are much inflamed but do not suppurate; when the latter occurs it is due to a mixed infection (staphylococcus, bacterium coli, etc.).

The *disturbances of acute gonorrheal prostatitis* vary much in degree.

In the *lighter forms of catarrhal inflammation* the process extends from the posterior urethra into the prostate in the region of the caput gallinaginis, and provokes a more or less circumscribed area of inflammation. The urine becomes turbid and contains *comma shaped shreds,* which conform in outline to the ducts of the prostate glands. The prostate is not perceptibly enlarged.

A higher degree of acute prostatitis is presented by the *follicular form of inflammation.* The secretion is purulent, the ejaculatory ducts and the glandular body are swollen, and small follicular abscesses develop in the lumen of the prostatic glands, while the tissues surrounding them are tensely infiltrated with small cells.

In this event frequency of urination and pain are marked; the urine is strongly turbid. The prostate (on rectal palpation) is slightly enlarged and tender. Spontaneous recession of the process is the rule. Cicatricial obliteration of the ejaculatory ducts may be responsible for sterility.

The *treatment* is symptomatic.

The severest form of prostatitis is the *parenchymatous.* In this event the entire glandular tissue is saturated with exudate and is distended with small cell infiltration, so that it becomes two or three times its natural size, and the attendant disturbances are of a grave nature. Pain in the perineum is very severe and radiates into the rectum, the glans penis, the back and the thighs. Chills followed by high fever usually occur.

Palpation of the prostate is very painful. The organ feels hot, hard and tense; its surface is smooth. If the process does not recede in five or six days, abscess formation usually occurs. Suppurative venous thrombosis may lead to pyemia.

When an abscess forms, the rectal mucosa becomes edematous, soft, and friable. The prostate softens and pits on pressure. The subjective symptoms now reach their maximum; retention of urine is the rule. The pus either perforates into the urethra or rectum; in either event the symptoms immediately subside.

In other cases the process does not recede and the prostate remains in a state of *chronic inflammation,* attended with the formation of perineal and anal fistulae, which discharge urine mixed with pus. In not a few instances the process is attended with extensive urinary infiltration of the tissues, of which large areas undergo necrosis and establish communication between the rectum and the urethra. These cases are not amenable to treatment and gradually die from exhaustion and amyloid degeneration.

The *treatment* in the early stages consists in rest in bed, regulation of the bowels, hot sitz baths, and the use of anodyne suppositories. The bladder is emptied with the silk Mercier catheter.

When pus forms, the abscess must be promptly opened. If the abscess lies close to the rectum and the mucosa of the latter is in-

volved in the process, the opening is made in this situation. When the abscess is located deeply, the prostate is systematically exposed through the perineum and drainage is established. The technic of approach is similar to that of perineal prostatectomy. The danger of rectoprostate fistula in the former method is great.

Chronic prostatitis in sequence to the acute form is unfortunately very common. The condition is indicated by persistence of the acute symptoms in moderate degree.

In the prolonged observation and treatment that these cases are subjected to by the urologist, it is important to remember that abscesses of considerable magnitude may develop without fever and other symptoms indicative of the true state of affairs, and that drainage of the prostate as for acute abscess is likely to be followed by relief.

Large chronic abscesses may be palpated. Smaller, suppurative areas may be recognized by means of the *three glass test.* The first glass contains the secretion of the urethra, the second that of the bladder; the prostate is then emptied by massage and the third glass contains the secretion from the suspected organ. A connection between chronic prostatic disease and certain neuroses (prostatic neurosis) seems to be established.

Tuberculosis of the prostate occurs most often in patients between twenty and forty years of age; as a rule, it is a part of tuberculosis of other portions of the urogenital system (73 per cent). The occurrence of isolated prostatic tuberculosis seems to have been established by Klebs.[4] This is no doubt hematogenous in origin.

Secondary prostatic tuberculosis usually extends from the epididymis and the vesiculae seminales; occasionally from the kidney (Schlange[5])

At first, the prostate is studded with small nodules of tuberculosis, which gradually coalesce, and, finally, the entire gland undergoes caseation and liquefaction. Not infrequently only one half of the gland is invaded, and this may undergo spontaneous healing. In other instances, the process extends to contiguous tissues and is responsible for periproctic and proctovesical tuberculosis, with multiple fistulae.

Early in the disease no characteristic symptoms of the disease are presented. Later, a urethral discharge containing tubercle bacilli may indicate the presence of the affliction; this is attended with painful micturition.

The *treatment* is modified in accord with the symptoms. Strange to say, most cases ultimately heal spontaneously. Total prostatectomy

has been followed by relief; it is rarely indicated. In all events, rectal drainage is to be avoided.

Lues and actinomycosis of the prostate are not often met with, or, at least, not often recognized. Schlange[5] quotes Jungano and Cohn in this connection. See also bibliography, No. 2.

Hypertrophy of the Prostate.— Hypertrophy of the prostate finds its anatomical expression in an increase in volume of the tissues of which normally it is constructed; a new formation of heterogeneous elements does not occur (Schlange[5]).

The *etiological factors* of prostate hypertrophy are numerous. Aside from *disposition* (heredity, gout, rheumatism, lues, tuberculosis), a number of local processes are held responsible for the condition. These are chronic prostatitis, gonorrhea, vesical calculi, strictures, hemorrhoids, excesses in *vino et venere,* obstipation, sedentary habits, etc. The contention that the condition is due to arteriosclerosis has been disproved. In any event, the disease is essentially one of old age.

Young,[1] basing his conclusions on the examination of 120 specimens, distinguishes three types of prostatic hypertrophy, (1) glandular, 100 cases; (2) fibromuscular, 14 cases; (3) inflammatory, 6 cases.

In the *glandular form* the surface of the gland is usually lobulated and of a soft consistence. On section, the organ may be more or less spongy (owing to dilated glandular acini) and studded with gaping orifices of small retention cysts, from which abundant secretion oozes. Usually, however, section of the gland presents numerous spheroidal tumors of different sizes, separated from one another by bands of tissue of denser character and various degrees of thickening.

In the *fibromuscular type* the consistency is much firmer, but it never has the induration of prostatic carcinoma. On section it is less succulent and distinctly more homogeneous in appearance, and is studded with isolated spheroids which may be almost or completely composed or fibrous or fibromuscular tissue. Occasionally, dilated orifices of the acini and retention cysts are seen. The gland rarely attains the size of the glandular type.

In the *fibrous or inflammatory type,* on gross examination, no spheroids are present, the cut surface being homogeneous or fibrous. The prostatic obstruction is the result of inflammatory processes, which produce a fibrous hyperplasia about the vesical orifice or result in the formation of an inflammatory bar. This type of gland shows very

little enlargement, but produces partial or complete retention of urine.

The hypertrophied gland may undergo malignant degeneration. The mechanical changes produced by enlargement of the gland are very important. The usual prostatic enlargement consists in the growth of an increasing number of spheroids surrounded by an artificially produced thickened capsule, leading to pressure distortion of the urethra in various ways as the masses increase in size.

In the simplest form there is enlargement of one lobe only, causing the urethra to deviate to the opposite side. More commonly both lobes are enlarged, which flattens the urethra anteroposteriorly. Later, the enlarged lobes project into the bladder to a great extent, at times extending upward to the symphysis; in these cases the urethra lies external to the growth. This must be taken into account in enucleating the glands, so as to avoid the sphincter during the manipulations.

FIG. 1015.—ENCAPSULATED OR ADENOMATOUS FORM OF PROSTATIC ENLARGEMENT.

Cross section (natural size) (Wallace).

The lateral lobes are most often affected. Median enlargement occurs in front of the ejaculatory ducts and beneath the urethra close to the vesical orifice. In advanced hypertrophy of this sort, the urethra assumes a slit-like or inverted crescent shape. Eventually the middle lobe forms a transverse bar, or it obtrudes itself into the vesical orifice as a round mass (sometimes as large as an orange) and acts like a ball valve, the mucous membrane lying in front and the vesical sphincter behind. As time goes on the lateral and median lobes are compressed by the vesical sphincter, forming sulci of varying depths; these are of diagnostic significance in the cystoscopic picture.

Hypertrophy of the median lobe of the prostate may occur without lateral enlargement. However, this, like enlargement of the anterior commissure of the prostate, is very rare.

The obstruction to micturition, caused by hypertrophy of the gland, is responsible for increased expulsive effort on the part of the bladder, which becomes hypertrophied and later sacculates. As the urine stagnates in the latter, cystitis develops and this leads to renal complications — usually uro- and pyonephrosis.

The *symptoms* of prostatic hypertrophy present themselves in a fairly definite circle, as follows: (a) the period of urinary disturbances — increased frequency and difficulty of micturition; (b) the second period of chronic incomplete retention of urine, without distention of the bladder, and generally with some contraction of the organ; (c) the third period of incomplete retention with vesical distention and often intermittant complete retention of urine, requiring the use of the catheter; (d) the fourth period of continuous complete retention of urine, in which catheter life is established.

The patient at first finds difficulty in micturition, especially at the beginning, the stream being projected with less force and, at times, being smaller in caliber. Soon increased frequency develops; this is especially noticeable at night. Moderate pain and a sensation of weight and fullness about the perineum are complained of; tenesmus and even hernia may be induced by straining. Presently, a certain amount of residual urine remains after each urination; the vesical muscles through hypertrophy begin to lose their power and become atonic. Finally, well marked distention and atony ensues and, if neglected, "dribbling" occurs. This is followed by decomposition of the urine, which produces cystitis and increases vesical irritability, and eventually, the symptoms of uronephrosis and pyonephrosis are added to the picture and the general health is gradually impaired by the constant irritation and the absorption of toxic products.

Occasionally the early symptoms pass unnoticed or are looked upon by the patient as a sign that his kidneys are being freely flushed, and not until exposure or alcoholic excess produces an acute retention is the patient made aware of his condition and its serious aspect.

The *diagnosis* is based on the history and the urethral and rectal examination. Median enlargement may escape recognition by these methods. In this event the cystoscope readily makes the condition clear.

The *non-operative treatment* of prostatic hypertrophy includes general hygienic measures, diet and medication. These need not be discussed here. The use of the Röntgen ray and of radium have not

proved of value and, in some instances, have been followed by severe reaction. Perhaps "Röntgen castration" possesses some virtue (Wilms and Posner[6]).

Catheterization for the relief of urinary retention due to prostatic hypertrophy may be a life saving measure. In the hands of the skilled attendant its employment prevents the serious complications of the affliction. On the other hand, its improper use is likely to be responsible for serious infection of the urinary passages. In most instances the elbowed catheter, which can be boiled, shown in Fig. 1016, answers the purpose. In many cases the bladder need not be emptied more than twice daily. In not a few cases it is impossible to gain access to the bladder, except with the metal catheter; this is always a dangerous maneuver. If this is not readily attained, *suprapubic puncture* of the bladder is permissible. While it is true that carefully executed catheterization permits of a comfortable existence, it is also true that early operative measures of relief directed toward removal of the obstruction to urination are less likely to be followed by a fatal outcome than when the development of complications (especially infection) is awaited.

FIG. 1016.— MERCIER CATHETER.

The operative procedures are divided into the palliative and the radical.

Palliative operative measures have been gradually crowded into the background. They consist in *suprapubic cystostomy* and *vesicopexy*. The latter aims at the extraperitoneal fixation of the bladder at a high point, so that it will drain through the urethra. One is compelled to admit that the measure has occasionally succeeded in relieving the symptoms.

Measures designed *indirectly to influence* the growth of the prostate and to cause it to shrink have found little support in practice. They consist in simultaneous ligature of both internal iliac arteries, castration, sclerosing injections into the testicle and epididymis, and vasectomy. See bibliography of No. 2.

Operative measures which *directly attack the prostate* have met with better success.

Perineal prostatotomy, i. e., incision of the prostate through the median external urethrotomy incision, as practiced by Harrison, who left a large drain in the bladder for several weeks following the operation, was soon abandoned for Bottini's[7] galvanocaustic diaresis. An

exceedingly ingenious water cooled instrument was introduced into the enlarged prostate by way of the urethra, and a furrow burned into the enlarged gland. The measure did not accomplish the intent. Later the technic of the procedure was modified and improved by Freudenburg. The method enjoyed a temporary fervor. However, the not infrequent unfortunate accidents attendent upon the procedure soon led to its abandonment. A complete résumé of the various steps leading to modern prostatectomy may be found in the works of Young,[1] Socin and Burkhardt,[2] and v. Frisch.[3] Two methods of removal of the prostate will be described here.

Suprapubic prostatectomy was first performed by Amussat in 1827. In 1888, McGill[9] reported 24 cases. Fuller,[10] of New York, in 1894, made the first complete suprapubic prostatectomy performed in America. Five years later, Freyer[11] described the operation as follows: "A stiff gum catheter is inserted into the bladder, which is then washed out and left distended with boracic acid solution; the nozzle of the syringe employed for this purpose, and which is filled with solution, is inserted into the end of the catheter, thus acting as a plug to prevent leakage from the bladder, the syringe being ready to further distend the bladder with fluid if necessary, as the operation proceeds. An incision varying in length from two and one half to three and one half inches, according the structures of the patient and the size of the prostate, is made in the median line of the abdomen and a vesical incision about an inch long in the vertical direction toward the symphysis. On withdrawal of the scapel the forefinger is introduced into the bladder, and if any calculi are present they are at once removed by forceps or scoop. The forefinger of the other hand is then introduced into the rectum to render the prostate prominent in the bladder and to keep it steady during the manipulations by the first hand. The mucous membrane over the most prominent portion of one lateral lobe or over the so-called middle lobe is scored through by the sharpened fingernail and gradually detached by it from the prominent portion of the prostate in the bladder. This portion of the enlarged prostate is covered merely by mucous membrane, so that when it is scraped through and detached, the true capsule is at once reached. Keeping the finger's point in close contact with the capsule, the enucleation of the prostate out of the enveloping sheath outside of the bladder is proceeded with by insinuating the finger tip in succession behind, outside, and in front

of one lateral lobe, thus separating the capsule from the sheath. The finger is then swept in a circular fashion from without inward, in front of and to the inner side of the lobe, detaching this from the urethra, which is felt covering the catheter, and is pushed forward toward the symphysis between the lateral lobes, which will, as a rule, have separated along their anterior commissure in the course of the manipulation. The other lobe is attacked and treated in the same manner. The finger is next pushed well downward to the prostate and the inferior surface of the gland is peeled off free within its sheath and separated from the urethra. With the finger in the rectum, aided by that in the bladder, it is pushed into the bladder through the opening into the mucous membrane, which, during the manipulations, will have become considerably enlarged. The prostate, which now lies free in the bladder, is withdrawn by strong forceps through the suprapubic wound. There is, as a rule, very little bleeding from the operation. The cavity left by the removal of the prostate disappears, owing to the inherent elasticity of the sheath, the contractility of the surrounding muscles, and the pressure of the pelvic structures. The contractility of the cavity will be greatly facilitated by pressing its apposing surfaces together by the points of the fingers in the bladder and rectum. Irrigation of the bladder by a hot lotion through a

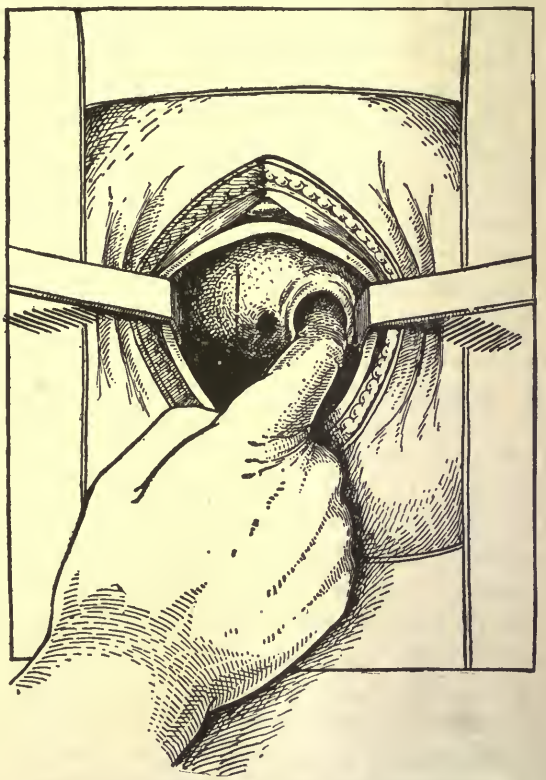

FIG. 1017.— TRANSVESICAL PROSTATECTOMY.

catheter and out by the suprapubic wound will help to check the hemorrhage, but if irrigation is continued for more than two minutes it increases the bleeding. The bladder having been cleared of clots, while the irrigation is still proceeding, a rubber drainage tube seven eighths of an inch in diameter and about five inches long, and provided with large lateral openings, is introduced into the bladder for a distance of not more than one inch. Nothing is introduced into the prostatic cavity, the object being to facilitate its contraction. The edges of the parietes are now brought together around the tube by silkworm gut sutures, some of which should pass deeply through the recti muscles, leaving room for a small perivesical gauze wick. No sutures are inserted into the bladder. Before withdrawing the catheter and applying the dressings, the bladder is once more irrigated to ascertain that the drainage is free. During the 24 hours after the operation there will generally be some clots lying in the drainage tube; these should be removed by long forceps at each dressing. The bladder should be irrigated daily, using a long glass nozzle, which is introduced into the drainage tube, care being taken not to cause pressure in the bladder, which might disturb the prostatic cavity. The tube is removed in from three to five days. Irrigation of the bladder must be continued once or twice daily, the fluid being allowed to flow out through the suprapubic wound around the nozzle, until the fistula contracts, when the irrigation is accomplished by alternately filling the bladder and withdrawing the nozzle, thus allowing escape of the fluid. As the case advances more and more pressure on the bladder may be employed. After nine days, irrigation of the bladder by means of hydraulic pressure through the urethra may be employed, but when natural urination is established no more irrigations are used. The management of the bowels is of the utmost importance. For three or four days previous to the operation they should be moved freely once daily by means of a laxative pill given at night and a mild saline in the morning, and on the morning of the operation by means of an enema. The bowels should then be left undisturbed for two or three days, and then they should be moved freely by castor oil; after this they should be moved gently once a day. Secondary hemorrhage has occurred in a few instances, and, should it persist and give rise to pain and spasm from the accumulation of clots in the bladder, no time should be lost in reopening the suprapubic wound and inserting a large drainage tube for three days, to relieve the pressure on the walls of the prostatic cavity."

Although partial prostatectomy by the perineal route was previously performed by a number of surgeons, *complete perineal prostatectomy* was first accomplished by Goodfellow[12] (1891) who succeeded in removing both lateral and a median lobe by this route. The operation is described as follows: With the patient in the ordinary lithotomy position, the legs held by an assistant, the bladder being empty or full as the case may be, the lithotomy staff is introduced, the legs elevated somewhat, a median incision from the base of the scrotum to the margin of the anus is made through the bulbous and carried to the membranous urethra, which is entered with a straight lithotomy knife and the opening extended into the bladder. The finger is then introduced into the bladder, the staff removed, and the moderate flexion of the legs on the abdomen and thorax increased to as great an extent as possible. Then, with the hand on the hypogastrium, the bladder is depressed and the enucleation, beginning at the beak of the prostate below and working upward next to the bladder, or from above on either side downward, is carried out; the time consumed for complete enucleation is between five and ten minutes. The method requires considerable skill.

Young[13] presents an exhaustive study of the perineal method.

Cancer of the prostate is not uncommon. In a series of 65 cases of prostatectomy, Wallace[14] found 9 to be carcinomatous, while Young[1] found a similar condition of affairs in 68 out of 250 cases. As in carcinoma elsewhere in the body, the process may be diffuse or circumsribed.

The *clinical picture* is, at first, that of hypertrophy of the prostate, but as the process progresses much more rapidly the symptoms are correspondingly more marked. Hematuria is likely to occur at almost any stage of the disease. On rectal palpation the prostate is hard and more or less firmly fixed to the contiguous tissues. Extension of the disease to the rectum and bladder is characterized by symptoms referred to these organs.

The *treatment* is similar to the operative treatment of hypertrophy. In many cases only the palliative measures (cystostomy) are indicated.

Sarcoma of the prostate is very rare; the diagnosis is possible only by examination of an excised sector of the growth. The rapid development of a large tumor of the prostate in a young person would justify the suspicion of sarcoma.

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CHAPTER IV

SURGERY OF THE PENIS AND URETHRA

Congenital Malformations.— Webbed penis is a very rare congenital deformity. The inferior surface of the organ may be partially attached to the scrotum, or the penis may be covered by the integument which covers the testicles, the glands alone protruding. The organ is usually underdeveloped. A hypospadias or a shortened corpus spongiosum frequently coexist. Plastic correction of the deformity is usually feasible, unless the penis is rudimentary.

Micropenis is usually associated with other genital deformities; by itself, it possesses no surgical importance. *Megalopenis* is not, as a rule, attended with other malformations. It is not amenable to surgical treatment. A few cases of *double penis* have been reported (Heller[1]).

Inflammations of the Penis.— In the *acute* variety as a rule only the skin and subcutaneous tissues are involved. Occasionally a low grade of inflammation attacks the corpora cavernosa; this form of infection usually occurs in connection with trauma, lues, gout, urethritis, and stricture of the urethra.

The *chronic* type of inflammation is also limited to the skin and subcutaneous tissues, usually in the region of the frenulum; it is very rare.

Aside from the usual *symptoms* of inflammation, this class of lesions of the penis is attended with difficulty and frequency in micturition and painful erections. The dorsal lymphatics are usually indurated and the inguinal glands swollen. The swelling of the penis is likely to be responsible for phimosis and paraphimosis.

Early liberation of exudate with the knife is the *treatment* of choice. This becomes imperative when suppuration is established. The tissues are incised in the direction of the length of the penis.

Gangrene of the penis occurs in connection with various *local* inflammatory processes, extravasation of urine, injuries, strangulation from phimosis and paraphimosis, and by foreign bodies caused to encircle

the organ by sexual perverts. The condition also develops in the course of acute infectious diseases, especially scarlatina and smallpox. It may be *dry, moist* or *diffuse.*

The *localized moist type* is usually preceded by the *symptoms* of inflammation. The organ becomes enormously swollen and discolored; the skin is bluish black in color and studded with greenish areas and vesicles. The constitutional reaction varies with the extent of the process.

In the *dry form of gangrene* the penis become edematous; soon purplish spots make their appearance. This is followed by the formation of a vesicle, which ruptures and discharges bloody fluid, leaving a dry, dark area.

The *treatment* of gangrene of the penis is primarily aimed at removal of the cause. This includes circumcision, liberation of extravasated urine, and removal of constricting agents. Liberation of exudate by free incision is permissible. In dry gangrene amputation is necessary.

Hypertrophy of the penis appears as a part of elephantiasis (Part V, chap. iv).

Injuries of the Penis.— Subcutaneous laceration of the corpora cavernosa occurs when the penis is erect; it is frequently called *fracture of the penis.*

The condition is the outcome of violence and consists in a tear or rupture of the tense tunica albuginea, extending more or less into the corpus cavernosum. The blood contained in the latter is effused into the subcutaneous tissues and forms a hematoma, which, at times, extends from the prepuce to and upon the abdominal wall and may assume enormous proportions. Occasionally the urethra is torn (Rammstedt[2] collected 18 cases); this leads to retention and extravasation of urine with its attendant dangers.

The *treatment,* in cases of moderate degree, consists in rest of the organ. When the extravasation is extensive, the distended tissues must be freely incised. When the urethra is torn, perineal urethrotomy, which temporarily diverts the urine from the site of injury, is indicated.

Open injuries of the penis are usually attended with copious bleeding. Division of two thirds of the circumference of the penis may be followed by healing. Division of both corpora cavernosa with maintenance of the urethra and spongy bodies has also been followed by healing (Rammstedt[2]).

The *treatment* of wounds of the penis is similar to that of wounds in other situations. Complete arrest of bleeding and exact repair by suture are especially important.

Partial or complete avulsion of the skin of the penis is not an uncommon injury. Healing is very likely to be attended with grave cicatricial deformation of the organ and consequent interference with urination and coitus and painful erections.

In the *treatment* it is important to remove all crushed and devitalized tissue and to make immediate plastic repair of the denuded areas. When infection is present, it is, of course, necessary to await its recession before repair is made; however, complete cicatrization must not be permitted.

Fortunately the ample integument of the scrotum is well adapted for the purpose. If the scrotum is also injured and is not available, flaps from the inner side of the thigh or abdomen may be utilized with advantage. In either event the penis is placed beneath a bridge flap, which is later separated from its original position and caused to encircle the organ. In cases of partial avulsion of skin, the Thiersch method of grafting may be employed (Bessel-Hagen[3]).

Dislocation of the penis is a term applied to laceration of the skin of the penis, scrotum or abdomen, in which the glans slips through the opening and remains fixed in its new position. The mechanism of the injury is easy to understand. The elastic skin of the genitals is stretched by the provocative force and tears at one of the situations stated; when the skin is released, the penis slips out through the tear or, if the skin is not torn, the penis — not being as elastic as the skin — may bury itself in any one of the situations stated.

In *fresh cases* correction of the malformation is easily accomplished. In *old cases* it is necessary to dissect the penis out of the fibrous tissue with which it becomes surrounded in its new environment. In this event plastic measures may have to be resorted to (Kausch[4]).

Morbid Conditions of the Prepuce and Penis.— Phimosis may be defined as a condition of the prepuce which interferes with its retraction; when the latter is forcibly accomplished it is likely to be irreducible (*paraphimosis*). Phimosis may be congenital or acquired.

Congenital phimosis is likely to be responsible for a number of varied clinical manifestations. The primary disturbance consists in difficulty in urination. This is followed by congestion of the urethra and bladder and, later, by urethritis and cystitis. In some instances the phimosis is responsible for uro- and pyonephrosis.

The *symptoms* often bear resemblance to those of vesical calculus, including tenesmus and priapism. When the preputial orifice is very small urine collects within the prepuce (*sacculation of the prepuce*); the distention may be enormous (Fig. 1018). In these cases the urine dribbles and the glans is in a state of chronic inflammation (*balanitis*). A tightly adherent prepuce is also attended with balanitis, so that the foreskin becomes adherent to the glans. This leads to sexual excitement and the child is likely to masturbate. The condition seems to favor the occurrence of epithelioma later in life.

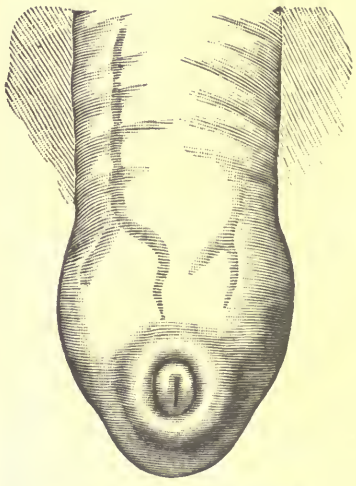

Fig. 1018.—Congenital Phimosis.

Acquired phimosis is due to cicatricial deformation following inflammatory processes.

The treatment of phimosis is taken up later.

Paraphimosis is a much more urgent condition than phimosis. It is due to a narrowed preputial orifice, which is forced behind the corona of the glans and strangulates the latter. The constriction is soon followed by great edema and congestion of the distal part of the organ, which may lead to strangulation necrosis. The attendant pain is severe and urination is interfered with.

The *treatment* consists in forcible replacement of the prepuce. This is accomplished by grasping the penis between the first and second fingers of both hands and compressing the glans with the thumbs; at the same time the prepuce is drawn forward (Fig. 1019). Failing in this, the operative procedure described farther on may be employed.

Fibrous sclerosis of the corpora cavernosa (cavernitis fibrosa) and *corpus spongiosum* is characterized by the development of fibrous tissue in the sheath, the septum or the erectile tissue of the penis. It follows an insidious course and makes its appearance without any discoverable cause. The lesion appears in any portion of the penis and may be unilateral or bilateral, single or multiple. In most cases a single nodule forms in the dorsum of the penis. This develops very

gradually and ultimately causes deformation of the penis during erection.

The *treatment* is unsatisfactory. Wälsch[5] claims relief following the injection of *fibrolysin.*

Fibrous and osseous transformation of the penis is a rare condition. Its pathogenesis is unknown. Extirpation of the process is permissible.

Tumors of the Penis.— Cystic tumors of the skin and subcutaneous tissues do not possess regional peculiarities.

Papillary excrescences of the glans and prepuce are very common. They are usually simple pedunculated warts, but occasionally develop into large cauliflower-like masses, which bear some resemblance to papillary carcinoma. They are readily ablated with the knife or scissors; the bases should be cauterized.

Fig. 1019.— Reduction of Paraphimosis.

Sarcoma of the penis is rare. It is usually primary and develops in the erectile tissue, where it grows very rapidly. The diffuse form of sarcoma quickly invades the inguinal and perineal glands. Melanosarcomata occasionally originate in the glans (Fisher,[6] Payr[7]).

The *diagnosis* is made by examining a sector of the growth with the microscope.

Early amputation gives what little hope there is for relief.

Carcinoma of the penis is not uncommon (3 per cent of all cancers observed in the male).

The *causative* influence of congenital phimosis and of psoriasis preputialis is generally accepted.

Küttner[8] divides the disease into three main types, (1) papillary cauliflower cancer; (2) carcinomatous ulcer; (3) non-papillary carcinoma.

Cauliflower carcinoma (Fig. 633) frequently occurs in connection

with congenital phimosis and develops on the glans and the under surface of the prepuce. The latter is soon perforated and the process follows its unrestrained course along the penis. When no foreskin is present, the growth begins on the glans in the form of a small nodule. As the destructive cellular invasion progresses the penis is gradually destroyed.

The *carcinomatous ulcer* presents all the characteristics of the process described in connection with carcinoma of mucous membranes (Part VI, chap. xvii).

Non-papillary carcinoma is very malignant. It bears a close resemblance to medullary carcinoma of the breast.

Carcinoma of the penis extends by way of the abundant lymph vessels of the corpora cavernosa, involving the inguinal lymph nodes first and soon reaching the retroperitoneal glands of the pelvis.

The *diagnosis* of carcinoma of the penis is based on consideration of all the factors extensively discussed in the general part of this work. In doubtful cases the question is decided by microscopical examination of a section of the growth.

The *treatment*, early in the disease, consists in *amputation* of the penis; in advanced cases *complete emasculation* is necessary. In the former, the inguinal glands, whether affected or not, are removed (Rumpel[9]).

Operations on the Penis.— Circumcision is performed by the clamp or dorsal incision method; the latter is preferable. The dorsal aspect of the prepuce is grasped on each side of the median line with mosquito clamps and the prepuce is slit to the corona. A suture is taken at this junction through skin and mucous membrane, acting as a tension suture; the redundant tissue is then cut away on each side, being sure to remove sufficient tissue on the under side to prevent the ugly projection below the frenulum so often seen. Bleeding vessels are clamped and ligated. Catgut sutures are then introduced laterally and at the frenulum; in the adult additional sutures between the above four sutures may be necessary. The sutures are tied and left long, and, between the stumps of catgut, gauze, immersed in vaselin, is tied.

In *irreducible paraphimosis* the constricting prepuce is freely divided with the knife.

Incurvation of the penis, if due to a constricting band, is subjected

to the following maneuver: An incision one inch in length is made in the median line, beginning just back of the glans, and dividing all the strictures down to the fibrous sheath of the corpus cavernosum. A wedge shaped portion of the latter is removed a few lines behind the glans. The edges of the corpus cavernosum are then approximated with buried catgut sutures and the skin closed.

If the incurvature is downward and associated with hypospadias, Duplay makes one or more transverse incisions upon the under surface of the penis, completely dividing the constricting bands and incising the corpora cavernosum as deeply as is necessary to meet the indica-

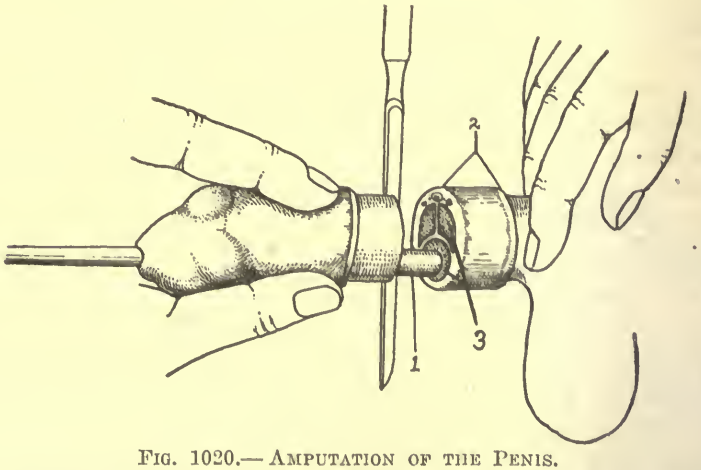

FIG. 1020.— AMPUTATION OF THE PENIS.

(1) The urethra being freed prior to section, (2) the reflected skin flap, and (3) the penile tissue cut through.

tions. Then the two extremities of the wound are united by means of fine silk sutures, after which the edges are approximated in the usual manner, the suture line being placed transversely.

In *amputation of the penis* the circular flap method is most frequently employed. A metal bougie is inserted into the urethra and the glans penis is pulled forward with catch forceps. A circular incision is made well back of the diseased tissue to the fibrous sheath of the cavernous bodies. The skin flap is freely dissected back so as to form a roomy covering for the stump. The cavernous bodies are next divided to the corpus spongiosum; the urethra is dissected free

from its attachment to the organ at least a quarter of an inch in advance of the point where the corpora cavernosa were divided, and the corpus spongiosum is then sectioned. Thus the corpus spongiosum is left longer than the corpora cavernosa. The tourniquet which was placed at the root of the penis is removed after ligature of the dorsal artery and vein. Only occasionally does the cavernous artery need ligature, although a gush of blood occurs upon release of the tourniquet. The urethra is split for at least one quarter of an inch on the floor of the canal; the skin flap is turned over the end of the stump and the urethra sutured to it, thus forming a spread out urethral orifice. A soft rubber catheter is inserted into the urethral canal and continuous drainage established until healing takes place.

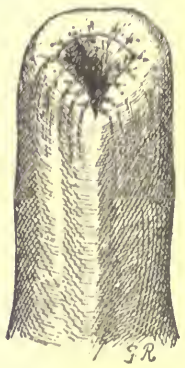

FIG. 1021.— AMPUTATION OF THE PENIS.

The skin pulled over the stump of the corpora cavernosa is united to the upper urethral wall transversely, and the split edges of the skin and urethra are united on each side, making a new meatus with a triangular opening.

Extirpation of the penis is usually performed for the relief of malignant disease. In all instances the inguinal glands are removed first. The penis is extirpated in the following way: The patient is placed in the lithotomy position and a urethral guide inserted into the bladder. An incision extending along the raphe of the scrotum from the root of the penis to the perineum separates the two tunicae with the testicles from one another, and exposes the corpus spongiosum in the bottom of the wound. The corpus spongiosum is now dissected away from the corpora cavernosa and the urethra freed, if necessary, as far back as the leaflets of the triangular ligament. The bougie is removed and the urethra severed and placed to one side for the present. A circular incision completely encircles the root of the penis, dividing the skin to the fibrous sheath. The skin flap is pushed back toward the pubes. The next step consists in division of the suspensory ligament and detachment of the corpora cavernosa until only the crura remain, which are cut in turn. Bleeding vessels are controlled and the skin closed by interrupted silkworm gut sutures. The urethra is split for one half inch on its under surface and fastened, as far

anteriorly as is possible, to the skin. A rubber catheter is inserted and left in the urethra until the seventh day.

Surgery of the Urethra — Method of Passing a Sound.— With the patient lying on his back and the glans cleansed, the lubricated sound is gently introduced into the ureters and *allowed to proceed by its own weight.* The shaft of the instrument is carried over the abdomen and is held in the median line (Fig. 1024). The beak is now at the peno-scrotal junction and entering the fixed part of the urethra. *At this time only is it proper to begin to elevate the shaft.* During the elevation the beak is made to describe the arc of a circle whose concavity is directed toward the pubes, thus following the curve of the fixed part of the urethra, the instrument nearing the bladder. While this occurs, the handle and shaft are following a curve in the opposite direction. To prevent the tip of the sound from hanging in the lower segment of the triangle produced by the natural bagging of the floor of the urethra in front of the triangular ligament, pressure is made with the left hand against the perineum and the beak is gently lifted over this possible obstruction. This introduces the beak into the membranous urethra. By depressing the handle of the sound between the thighs (Fig. 1025), as the inner end mounts the upward inclination of the prostatic urethra, the sound enters the bladder. By gently turning the instrument on its own axis one can be sure the sound is in the bladder.

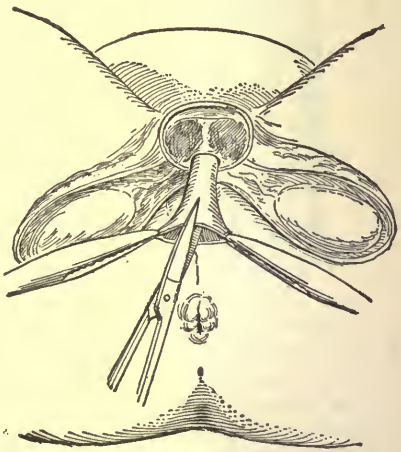

FIG. 1022.— EXTIRPATION OF THE PENIS.

After the penis is removed the divided urethra makes better union with the skin.

Urethroscopy visualizes the interior of the urethral canal. Of the various instruments obtainable, that of Guiteras, shown in Fig 1026, is as satisfactory as any (Keyes[10]). The instrument makes it possible to recognize lesions of the urethra and to apply appropriate local treatment. The most important lesions are the chronic inflammatory processes involving the lacunae of Morgagni, the verumontanum, etc., polypi, calculi, and foreign bodies. Occasionally a very small opening in a stricture may be located.

Malformations of the Urethra.— Hypospadias is a congenital defect of the anterior urethra, the outcome of arrested fetal development.

The *classification* of the various forms is based on the extent and character of the malformation and the position of the adventitious urethral opening, i. e., glandular or balanic; penile or scrotal.

Simple glandular hypospadias causes little discomfort. The prepuce is frequently malformed or may be absent.

In the *penile form of hypospadias* the urethral orifice may be situated at any point of the under surface of the penis from just back of the glans to the scrotal portion of the canal. When the urethra is intact as far as the glans, the penis is, as a rule, fairly well developed. The associated malformations are usually proportionate to the degree of urethral defect.

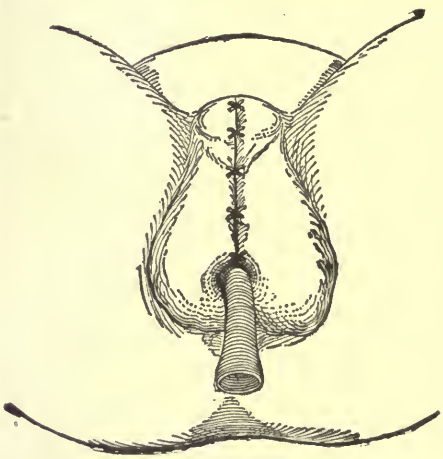

FIG. 1023.— EXTIRPATION OF THE PENIS.

The operation complete and a retained catheter placed.

In *scrotal and perineal hypospadias* the penis is likely to be underdeveloped; the glans may be broadened or curved downward. On lifting the penis a funnel shaped orifice lined with mucosa is discovered, corresponding to the deepest portion of the urethral outlet. Often a deep cleft divides the scrotum into lateral halves, each of which contains a testicle — if these have descended (Beck[11]).

The *treatment* consists in the performance of a series of plastic operations; good results can be obtained only at the expense of great perseverance. In the scrotal type, surgical treatment is of no avail. In children it is best to operate at about the age of four years.

The *curvature* of the organ, by freeing the scrotal adhesions, is first attempted, simultaneously restoring the glandular portion of the urethra. After recovery from this operation, perineal drainage is established and an effort made to restore the missing portion of the urethra, and, finally, the continuity of the latter is established by

closing the distal and proximal opening of the canal by means of flaps from the integument covering the penis and scrotum. Usually several operations are required to do this.

Beck[11] has devised an ingenious *operation for balanic hypospadias,* or where the urethra terminates slightly behind the glans. Instead of forming a new urethra, the one existing is dissected free, dislocated forward, and carried through an opening made in the glans, and sutured in place (Figs. 1027–1028).

Epispadias is a congenital deformity in which the entire roof or

Fig. 1024.— First Step in Passing a Sound.

part of the urethra is undeveloped. It occurs in several degrees: (a) limited to the glandular portion; (b) penile portion; (c) the glandular and a portion of the spongy urethra; (d) extending from the glans to the pubes. The impairment of function depends upon the type presented. Slight interference with coitus occurs in the glandular form, while the complete variety is attended with partial or complete incontinence of urine.

As the penis is rudimentary in the complete type, repair is impossible. The operation suggested by Duplay[12] probably gives the most

satisfactory results. At the first sitting the penis is freed from adhesions and the organ straightened. In the second step perineal drainage should be established as a preliminary measure. The septum between the corpora cavernosa is depressed by a glass rod and the bodies brought together; the cleft thus formed is utilized to form the floor and a portion of the sides of the canal, the roof being constructed by approximating the denuded margins of the corpora cavernosa by means of a modified quilled suture (Figs. 1029–1030). The sutures

Fig. 1025.— Third Step in Passing a Sound.

are removed on the seventh day, when the perineal drainage is discontinued and the space between the urethral orifice and the newly formed canal is closed by scarifying the edges of each, approximating the latter and holding them in position by silkworm gut sutures or modified quill sutures.

Traumatism to the Urethra.— Contusion of the urethra is not common; it is produced by direct violence and instrumentation. When the skin remains uninjured, and the urethra and other structures of

the penis are crushed, the outcome is likely to endanger the integrity of the entire organ.

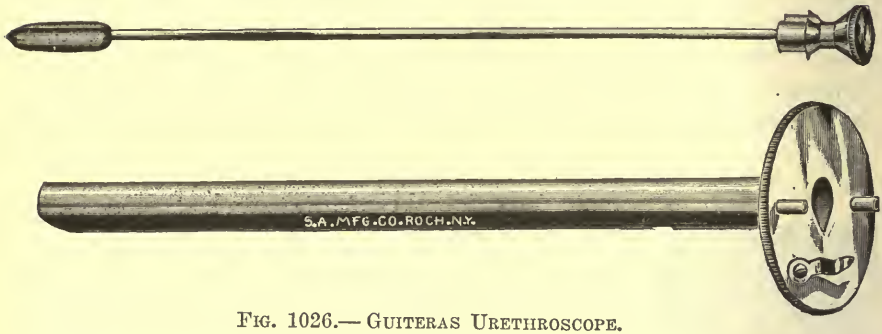

FIG. 1026.— GUITERAS URETHROSCOPE.

Injuries inflicted directly to the urethra usually occur in the form of a contused or lacerated wound, produced by foreign bodies, by calculi, or during careless instrumentation.

Rupture of the urethra is produced by violence or is due to pathological changes accompanying stricture. Traumatic rupture is usually located near the bulbomembranous junction; it may be associated with fractured pelvis, or caused by attempts at intercourse or rape, the turgid penis forcibly impinging upon the pubic bone of the female.

The *symptoms* are dependent upon the character of the injury. A simple contusion gives rise to a sore, bruised feeling at the site of injury, which is increased by walking, urination, or erection of the penis.

The disturbances consequent to *wounds* vary with the size and site of the injury. Pain, swelling, hemorrhage, difficult urination, and retention or extravasation of urine may occur. If the urethra is perforated the urine may flow through the open-

FIG. 1027.— BECK'S OPERATION FOR HYPOSPADIAS.

Second step, showing the flaps dissected to one side, the urethra freed and the incision up through the glans (from Keen's "Surgery").

ing. Swelling seldom occurs unless damage to the surrounding tissues, together with urethral rupture, takes place. Sudden onset of swelling that involves the perineum, scrotum and penis, associated with extravasation of urine, is diagnostic of ruptured urethra. Hemorrhage varies from a few drops to a severe flow of blood. From incised wounds in the anterior urethra the blood flows in a continuous small stream, being uninfluenced by urination. In partial rupture of the urethra only a few drops of blood may appear; this is increased by urination, the blood preceding the urine.

FIG. 1028.—BECK'S OPERATION FOR HYPOSPADIAS.

The urethra pulled through the glans (from Keen's "Surgery").

Moderate hemorrhage may cease spontaneously; in this event the clots may be discharged upon urination and bleeding may recur, the blood preceding the urination. In injury of the deep urethra, blood also precedes urination. However, if retention develops, the blood flows back into the bladder, mixes with the urine and is discharged with it, often in a bright red stream. In injuries of the urethra, unless the latter is ruptured, urination is seldom interfered with until twenty-four hours after the injury; by this time the spasm caused by the development of inflammation and the attendant edema of the mucosa narrows the lumen of the urethra, making urination difficult or impossible. Pain is not generally a troublesome symptom.

The *treatment* of simple contusions consists in the application of a cooling lotion, and suitable medication which renders the urine bland and allays sexual excitement. In wounds, the indications are to control hemorrhage, relieve retention of urine, when present, the prevention of extravasation by early perineal drainage, and relief of pain. An external wound communicating with the urethra is sutured and continuous drainage established by means of a retention catheter, until union has occurred.

Extravasation of Urine.— Urinary infiltration always indicates partial or complete *rupture* of the urethra. Its causative factors are straining in cases of retention where the walls of the urethra have undergone pathological change, or it may occur central to a calculus or foreign body. Careless instrumentation is not rarely responsible for rupture of the urethra. It is most commonly encountered in a stricture of small caliber or as a result of traumatism.

The *symptoms* are local and, later, constitutional. A partial rupture of the mucosa and a few muscle fibers is not attended by extravasation. In case of trauma behind a stricture, partial rupture of the floor of the urethra permits of a small, localized infiltration of small amounts of urine at a time into the periurethral tissues, which become filled with plastic lymph exudate. If the urine is sterile, a blind fistula forms and a well defined fibrous mass may be palpated at the site of the injury. When associated with strictures of small caliber, near the bulbomembranous junction, the plastic exudate increases and accumulates between the testicles in the scrotum or on one side of the perineum and scrotum. If infection occurs, the mass becomes inflamed and painful, increases in size, and finally ruptures, frequently leaving a fistula.

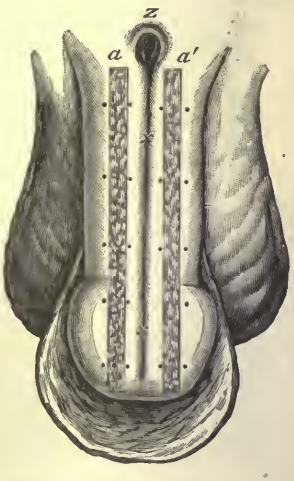

Fig. 1029.— Duplay's Method of Operation for Epispadias, Second Stage.

a a, Freshened surfaces; *z*, fistulous opening.

The rarest, and at the same time the gravest, form of *extravasation occurs in a rupture involving the corpora cavernosa,* the urine often dissecting forward to the glans, which may become gangrenous.

A rupture of the pendulous urethra is attended with extravasation over a large area, involving the common fascia of the organ and, finally, reaching the scrotum.

In *penile rupture with extravasation,* early ulceration of the skin may provide drainage before extensive damage to the tissues results.

In *rupture of the urethra* between the *attachment of the scrotum* and the *anterior layer of the triangular ligament,* the extravasation of urine manifests itself, first, as a tumor in the upper portion of the perineum and lower portion of the scrotum. As the swelling gradu-

ally extends the entire scrotum is invaded and the urine slowly dissects its way up on the pubes and abdomen.

Extravasation consequent to a *ruptured membranous urethra* first infiltrates the perineum, and eventually, if the *anterior leaflet of the triangular ligament* gives way, the infiltration is similar to that in which the bulb is involved; if the *posterior leaf* gives way, the urine infiltrates as when the prostatic urethra is perforated. Usually the urine extends along the course of the rectum, the tumor appearing in the anal region. Finally, the extravasation may infiltrate the subperitoneal connective tissue, giving rise to a pelvic cellulitis or causing peritonitis; occasionally it extends around the prostate and neck of the bladder and gains access to the space of Retzius.

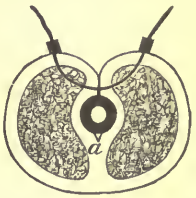

FIG. 1030.—DUPLAY'S METHOD OF OPERATION FOR TREATMENT OF EPISPADIAS.

Completion of second stage; *a*, median incision.

The *constitutional symptoms* are grave when rapid extravasation occurs. If the urine is infected, gangrenous cellulitis with constitutional symptoms of septicemia develops. Marked extravasation with a rapid wiry pulse, shallow respiration increased in rate, irregular fever, dry fissured tongue, and delirium rapidly occurs, and finally coma ensues.

The *treatment* — in gradual infiltration from a rupture behind a stricture with a peri-urethral inflammatory exudate, blind fistula or abscess — consists in the restoration of the caliber of the urethra by a suitable operation. Abscesses are incised and drained; fistulous openings are closed by plastic procedures. Continuous catherization is generally all that is required in partial rupture. Septic symptoms and signs of urinary infiltration demand perineal drainage.

Rupture of the urethra at any point from the scrotal attachment to the posterior leaflet of the triangular ligament is a grave surgical condition and calls for heroic treatment; this consists in the establishment of an external perineal urethrotomy to restore the normal caliber of the urethra and to provide free drainage of the infiltrated tissues. The *constitutional* treatment is supportive.

Foreign Bodies in the Urethra.— A foreign body in the urethra is usually inserted or forced through the meatus and may ascend through the urethra as the result of muscular contractions and erections, until it enters the bladder where it is forced back into the urethra and causes obstruction. A calculus may descend from the bladder or, in rare instances, may form behind a tight stricture.

The *symptoms* of the passage of small stones along the urethral canal are those of pain and discomfort. Partial or complete retention of urine occurs in accord with the size of the body. If ulceration of the mucosa has occurred, bleeding may be brisk. Infection causes urethritis and peri-urethritis; the latter often results in abscess. The symptoms are not severe, unless the body is located in the deep urethra — except when the pendulous urethra is obstructed. In this event the penis becomes enormously swollen and edematous. The symptoms of retention of urine are added when the canal is completely occluded.

The *treatment* consists in removal of the foreign body. If the body is small and round and smooth it may be passed spontaneously. This is aided by splitting the meatus and carefully dilating the distal lumen of the canal. The injection of sterile oil facilitates ejection of the body. Occasionally a body may be extracted with slender forceps introduced through the meatus.

If a stone is lodged in the deep urethra, it is best pushed back into the bladder and removed by lithotrity. Impacted bodies are removed by external perineal urethrotomy.

Pins, needles, and hairpins introduced with the sharp end pointing toward the meatus present difficulties. For the removal of a pin, Poulet[13] suggests the following:

"The head of the pin is firmly fixed with the thumb of the left hand; the penis is next bent at the part of the urethra corresponding to the point of the pin, which, in consequence, protrudes through the walls of the canal, and is withdrawn until the head comes in contact with the floor of the urethra. The shaft of the pin is now drawn down toward the root of the penis, and then, by pushing upward, the head of the pin emerges from the meatus and is withdrawn." If the problem presents difficulties the urethra is sectioned and the body removed; this should be done before infection develops.

Urethral calculi are composed entirely of phosphate of lime; they may be single or multiple and vary greatly in size. In long standing cases the stone either becomes partly embedded and fixed in the urethral wall, or forms a diverticulum which may contain numerous stones. In these cases urethral fistula is common, especially if a tight stricture exists. Peri-urethral abscess and rupture with extravasation are late complications.

The *symptoms* of a urethral calculus formed *in situ* simulate those of a stricture of large caliber. If impassable stricture exists, palpation or X-ray determines the presence of the stone. If the stone is em-

bedded or lies in a false passage, there is little urinary obstruction. In the penile urethra, stones are readily detected by palpation. A stone in the posterior part of the urethra may be revealed by rectal examination. In all doubtful cases the Röntgen ray should be employed. A stone searcher is used when there is little or no obstruction, i. e., when the stone is embedded and only partial obstruction exists. Endoscopic examination will generally reveal the presence of small embedded stones.

When a calculus is either held fast behind a tight stricture or encysted, the concretion can be removed safely only by one of the following operations: meatotomy, external urethrotomy, or perineal urethrotomy, depending upon the location of the stone.

Inflammation of the Urethra.— Inflammation of the urethra (male) may be specific or non-specific; acute, subacute or chronic; it may be anterior or posterior, or involve the entire canal.

The *non-specific* variety is rare compared to the specific. It runs a mild course and is amenable to treatment in a few days. The exciting factors are the ingestion of certain irritating foods and drugs, diabetes, rheumatic and gouty diatheses, irritating injections, sexual excesses, etc. The main etiological factor is trauma, produced by strictures, stone, foreign body, and abnormal conditions of the urine. It may be secondary to inflammatory lesions outside the urethra.

Specific urethritis may be due to syphilitic lesions, chancroid, tuberculosis, typhoid, pneumonia, diphtheria, and the pyogenic organisms, and, finally, the gonococcus, which is the most important of all and is discussed under a separate heading.

The *symptoms* are similar to those of gonorrhea, but generally of a much milder type.

Gonorrhea, as a whole, is taken up in Part II, chap. x. Its complications are discussed in connection with the surgery of the various sectors of the genito-urinary system. However, the most important disturbances are enumerated. They are classified as due (1) to direct extension; (2) to traumatism; (3) to general absorption of the disease.

Direct extension of gonorrheal urethritis is responsible for:

Balanitis, which is especially troublesome in cases of phimosis.

Lacunar abscesses caused by gonorrheal or mixed infection of one or more of the lacunae. The condition is characterized by a tense, painful swelling along the floor of the penis. The process may rupture externally or internally, or both; in the last event a fistula results.

Incision and drainage, as early as possible, is indicated. The fistula is at first *treated* with the galvanocautery or silver nitrate, and, if it still persists, plastic repair becomes necessary.

Chordee is due to inflammatory infiltration of the corpus spongiosum or of one of the corpora cavernosa, so that when erection occurs the penis is bent toward the involved side. The local application of cold and the internal administration of bromids are soon followed by relief.

Inflammation of Cowper's glands may give rise to deep suppuration in the perineum, closely resembling acute prostatitis. It is treated in a similar manner.

Direct transmission of gonorrhea (by means of the discharge from the vagina) to the female rectum is not uncommon; the disease is occasionally transmitted in the male in the course of unnatural practices. The infection is manifested by the clinical picture of acute proctitis. The *diagnosis* is made with the microscope.

Gonorrheal ophthalmia occurs in infants and in the adult. In the former event the disease is usually bilateral, the eyes being infected during the passage of the fetal head through the maternal vagina (ophthalmia neonatorum); while in the adult the process is usually primarily unilateral. In the *adult* the infection begins with redness and irritation of the conjunctiva. This is followed by a mucous discharge, which soon becomes purulent. The eyelids are red and swollen and the conjunctiva is much thickened and edematous. As the infection progresses, corneal ulceration, and even necrosis, and, still later, general panophthalmitis develop. The disease is the most frequent cause of blindness in children.

In *infants Credé's phophylactic treatment* is essential; it consists in instilling a drop of 2 per cent silver nitrate or of 25 per cent argyrol solution into each eye immediately after birth.

If the disease is not aborted and only one eye is involved, the normal organ is treated with a prophylactic instillation and is then protected by means of a shield. The treatment of the infected eye must be carried on day and night; it consists in boric acid irrigations and the instillation of argyrol solution at regular intervals. The frequency of the maneuver depends upon the severity and progress of the infection.

Gonorrheal infection of joints is not uncommon. The process usually invades one joint at a time and occurs in the form of synovitis or arthritis (Part II, chap. x).

Gonorrheal scleritis is a rare inflammation of the deep subconjunctival tissues. It is characterized by subconjunctival redness and

tenderness of the eyeball. The *treatment* is limited to instillations of atropin.

The *vaccine treatment* has been used in the complications of gonorrhea of long duration, with seemingly favorable results. It is most effective after the acute symptoms of the infection have subsided.

Tuberculosis of the urethra is usually secondary to that of other portions of the genito-urinary tract. It is not common.

The *symptoms* are those of chronic urethritis; the disease may simulate the initial lesion of lues, or one of the forms of subacute periurethritis. Endoscopic examination and bacteriological examination of the discharge makes the *diagnosis*. The bacterial investigation must be carried out with observance of the precautions stated in connection with tuberculosis of the kidney, bladder, etc.

The *local treatment* is similar to that of tuberculosis of the bladder. It is not very satisfactory. In advanced cases the question of amputation of the penis arises.

Tumors of the Urethra.— New growths of the urethra are rare; they do not present regional peculiarities.

Papillomata appear in the form of caruncle, polypi, multiple papillary growths, and vegetations.

Primary *sarcoma* is practically unknown, while primary *carcinoma* has occasionally been observed. *Secondary carcinoma* occurs in connection with that of the rectum, bladder, prostate, and penis.

Diseases of Cowper's Glands.— Inflammatory involvement of Cowper's glands has the same *etiology* as that of urethritis. This has been discussed with the complications of gonorrhea. In the chronic variety the urethra may be normal, the "Cowperitis" resulting in a hypersecretion of mucus, which is responsible for a constant mucoid discharge from the meatus.

Tuberculosis is very rare and follows a tuberculous urethritis. The disease develops insidiously with a painless enlargement of the gland.

The *treatment* consists in resection of the infected area and subsequent irrigation with bichlorid of mercury, followed by an application of phenol and alcohol.

Tumors have been observed in the form of cysts and carcinomata. In carcinoma the treatment, as elsewhere, consists in early and complete removal of the process.

Stricture of the Urethra.— Lessening of the lumen of the urethral canal may be temporary or permanent, congenital or acquired.

The *temporary* form is either inflammatory or spasmodic in nature;

the organic form is the outcome of pathological changes affecting the structures making up the wall of the urethra.

The *congenital* variety is rare; it is usually situated near the meatus or at the bulbomembranous junction of the urethra.

An *acquired* stricture may be inflammatory, spasmodic or organic in nature.

Inflammatory strictures are usually temporary and are associated with acute urethritis and edema of the mucosa. The obstruction usually disappears as the process recedes. However, the cellular infiltrate may become organized and an organic stricture be formed.

Spasmodic stricture is due to contraction of the muscular fibers of the urethral canal, the accelerator urinæ or the constrictor muscle. It occurs in connection with a contracted meatus, hyperesthesia, inflammatory lesions of the urethra, and diseases of the rectum or neighboring structures. It is often a complication of organic stricture.

Organic stricture (Fig. 1031) is the most common form of the affliction. The lesion is a sequel of *inflammatory and traumatic cicatricial processes.* The latter follow severe injury with tearing of the urethra; the inflammatory type is usually sequential to chronic gonorrhea. Gonorrheal strictures are most often located at the bulbomembranous junction, but also form in almost any portion of the pendulous urethra; rarely, if ever, in the prostatic canal. Traumatic strictures are single; the gonorrheal may be multiple.

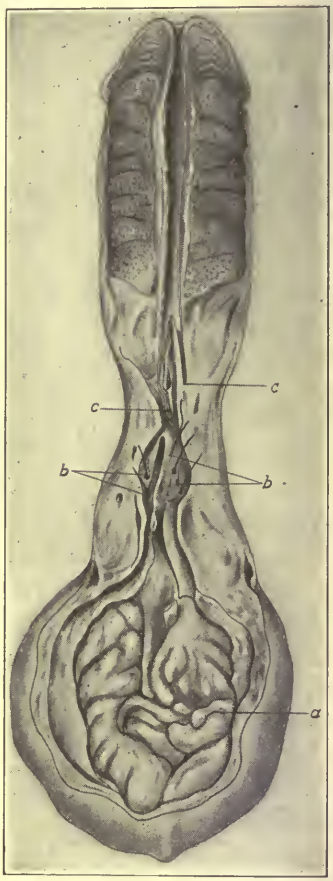

FIG. 1031.— STRICTURE OF THE URETHRA.

a, Diverticulum of the bladder; *b*, dilated excretory ducts of the prostate; *c*, false passages.

Strictures of the urethra are *classified* as follows: *bridle stricture* consists in a membranous bar stretching across the canal, narrowing

its lumen. An *annular stricture* encircles the canal. *Indurated or nodular stricture* is characterized by a thick, hard mass embedded in the corpus spongiosum at the site of the obstruction. *Recent or soft stricture* is one in which the peri-urethral infiltration has not yet organized. *Irritable stricture* is one in which pain and bleeding and urethral fever follows instrumentation. *Resilient or recurrent stricture* is one that contracts rapidly after dilatation or urethrotomy. The terms *permeable* and *impermeable,* applied to stricture, are self explanatory. *Cicatricial stricture* consists of a dense fibrous obstruction, at times cartilaginous in character. Traumatic strictures are generally of this type. *Centric stricture* is one where the opening is directly in the center of the coarctation. *Eccentric stricture* is one in which the opening is situated near the floor of the roof or laterally on either side of the median line. This type is much more frequent than the centric stricture. Strictures which do not admit of the passage of a number five (French scale) sound are termed *small caliber strictures.* Above this they are regarded as *large caliber.* Complete occlusion of the urethra is rare. The time required for the production of a stricture varies with the cause, those of gonorrheal origin developing very slowly, while the traumatic occur as soon as healing takes place.

Pathology.—An extensive peri-urethral cell infiltration usually exists in cases of subacute urethritis of long standing. This becomes only partially organized and causes slight contraction; it does not interfere materially with the dilatability of the urethra, but gives rise to congestion of the blood vessels in the affected area. This type of infiltration rarely becomes organized and the development of organic stricture is the exception. The plastic lymph is readily absorbed as the result of judicious urethral dilatation. A *cellular infiltration* in a localized portion of the urethral wall is more likely to undergo organization and become converted into scar tissue. The process is very gradual; usually a long time elapses before symptoms are produced. A stricture of this type is usually one of large caliber and takes years to produce disturbances. On the other hand, the canal may become rapidly contracted, scarcely admitting a filiform bougie, in a very short space of time. The *central portion* of the stricture consists of dense fibrous tissue; the margins are not highly organized and are frequently softer and more pliable. The affected area is occasionally the seat of erosions, ulcerations and papillary growths. As time goes on, the mucosa in the region of the stricture becomes hard and thickened, the superficial epithelium undergoes necrosis and, upon exfolia-

tion, makes its appearance in the urine in the form of characteristic threads of various sizes and shapes. The glands of Littré may be partially or completely destroyed. When the corpora cavernosa are extensively involved in the process, the urethra is obstructed from the glans to the bulb and is ultimately converted into a hard, rigid tube. Occasionally, in cases of long standing, the urethra central and distal to the stricture becomes thinned out and dilates. In the former event the dilated area is the seat of stagnated urine, which decomposes and is responsible for a low grade of urethritis associated with erosions and ulcerations. In the latter event the dilatation is the result of gradual atrophy and absorption of the strictured area. The back pressure coincident to persistence of stricture is finally attended with secondary changes in the bladder, the ureters, and the kidneys.

The *complications* of stricture of the urethra are likely to be very serious in character. Fenwick[14] has shown that a stricture of small caliber is always associated with more or less retention of residual urine. If ten ounces are retained, extensive changes in the ureters and kidneys always occur. Occasionally atony of the bladder results; this is especially so in hypertrophy of the prostate gland. More frequently, however, the obstruction is attended with vesical hypertrophy, which lessens the capacity of the bladder; this is regarded as compensatory in character. As the obstruction persists the bladder becomes sacculated; the saccules are often the seat of calculi. The mucosa becomes much thickened and thrown into folds. Finally, the ureters are thickened and distorted and the kidneys become the seat of uro- and pyonephritis and multiple abscesses.

The *subjective symptoms* of stricture of the urethra are *change in the character of the urinary stream,* frequency in micturition, dribbling of urine, tenesmus, retention of urine, and "ardor urinae." In some cases a chronic urethral discharge is present. Sexual irritability and atonic impotence may be associated with stricture of large caliber; aspermatism is likely to attend those of small caliber situated in the bulbomembranous urethra. Eventually the *character of the urine* changes and the coincident pathological processes in the bladder, ureters, and kidneys become responsible for *constitutional disturbances.* These are urinary fever, septicemia, uremia, and, at times, suppression of urine. On the other hand, multiple strictures may exist for a long time without causing disturbances; nor do all of those mentioned necessarily develop.

The *diagnosis* may usually be based on what Guyon calls the

"urethral history" of the case. A bacteriologic, chemical, and microscopic examination of the urine will demonstrate the character of the infection and also reveal the nature of the changes in the kidneys and bladder. By direct examination of the urethra, the location, caliber, and number of strictures is definitely established. The instruments required for locating the stricture and determining its caliber are conical steel bougies, bulbous bougies (*bougie á boule*), filiform bougies, an Otis urethrometer, Gouley's tunneled catheter, and, finally, the endoscope.

The introduction of a conical steel sound into the urethra, until it meets resistance, readily diagnoses a stricture of small caliber. The location of the obstruction is determined by grasping the shaft of the instrument at the meatus and withdrawing it; the distance between the finger and the tip of the bougie indicates the relationship of the stricture to the meatus.

By passing a number of bougies, from number sixteen (French scale) down, the caliber of the stricture is determined. If this is not

FIG. 1032.—WHITE'S SCALE.

Circumference	of	penis,	3	inches.	Caliber	of	urethra,	26-28	mm.
"	"	"	3¼	"	"	"	"	29-30	mm.
"	"	"	3½	"	"	"	"	30-32	mm.
"	"	"	3¾	"	"	"	"	32-34	mm.
"	"	"	4	"	"	"	"	34-36	mm.

feasible the urethroscope is inserted to the obstruction and a filiform guide is passed through the orifice in the stricture.

In locating the seat of large calibered strictures, the Otis urethrometer or a flexible bulbous bougie may be employed. There are four points of constriction in the urethra which in a bougie examination by the inexperienced are likely to be mistaken for stricture; they are from before backward; just behind the meatus; at a depth of about four inches from the urinary outlet; at the bulbomembranous junction, and at the junction of the membranous and prostatic urethra.

At a first examination it is sometimes impossible to differentiate urethral obstruction due to spasm and one caused by an organic stricture. Either condition may exist alone, but they are frequently associated, the spasmodic stricture occurring as a complication of the organic. At times, a spasmodic stricture may be passed if the bougie is held firmly against it, the spasm gradually relaxing and allowing the bougie to proceed. However, it may persist and the condition be

mistaken for a filiform stricture. Occasionally, if a second examination is made, the bougie passes without difficulty.

In strictures of large caliber a flexible *bougie á boule* is introduced into the bladder. The size of the instrument is determined by referring to the table shown in Fig. 1032, which gives the relation between the circumference of the middle of the body of the flaccid penis and the caliber of the urethra. If, on withdrawing the instrument, an obstruction is encountered, the point on the shaft at the level with the meatus is noted and the distance between this point and the bulb will indicate the site of the lesion. Care must be exercised not to confuse obstruction due to urethral spasm caused by pain or irritation with that due to organic stricture. In cases of organic stricture associated with spasm which cannot be passed by the various instruments, it is best to wait forty-eight hours before attempting to pass the stricture,

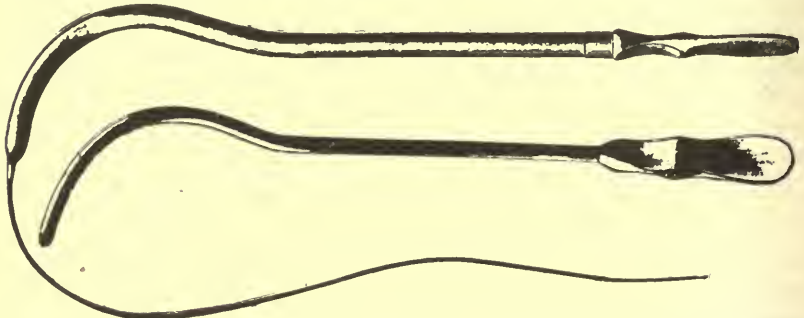

Fig. 1033.—Béniqué Sound, With and Without Filiform Guide.

except when an operative measure becomes imperative. The lower bowel is evacuated; a mixture of sodium bromid, belladonna and potassium citrate is administered, and, before instrumentation is again attempted, the urethra is anesthetized by a dram of 2 per cent cocain solution. This is followed by an injection of a dram of a 10 per cent emulsion of iodoform suspended in glycerine. After this, all symptoms of spasm usually disappear. Nitrous oxid narcosis may be employed instead of local anesthesia.

The *treatment* is based on the findings with regard to the caliber, resiliency, and irritability of the stricture, and the number of obstructions present. A general examination must not be omitted. In all instances the functional activity of the kidneys must be ascertained before operative measures of relief are attempted, except in emergency cases.

Great care and gentleness must be observed in all instrumentation. Preliminary *meatotomy* is frequently necessary before the introduction of instruments is possible.

Operations for Stricture.— Gradual dilatation is the method of choice in the treatment of the majority of strictures. It may be employed for years with every advantage to the patient. The measure is indicated in all cases of *recent dilatable stricture* in any portion of the urethra, and in those cases in which the stricture is not resistant, irritable or nodular. Firm, organized bands near the meatus or in the pendulous urethra are best treated by means of internal urethrotomy.

Dilatation of the stricture consists in the introduction of suitable catheters, sounds or bougies of a diameter which will just pass the stricture without incarceration. In most instances it is possible to pass a stricture with the French filiform bougie; these vary from a third to one and a half millimeter in diameter and are "resistingly elastic." It is at times necessary to introduce six or eight of these guides like the arrows in a quiver into the urethra before one of them passes the stricture.

In many cases permanent dilatation of the stricture is desirable. In this event a dilatating catheter is left *in situ* as in permanent drainage of the bladder.

Forced dilatation is feasible when a filiform cannot be made to pass the stricture. The instrument most frequently used for the purpose is the LeFort guide sound shown in Figure 1033. When the bougie has remained *in situ* for forty-eight hours, the stricture is usually sufficiently softened to permit of the use of the dilator. For this purpose the instrument devised by Kollmann[15] (Fig. 1034)—a modification of that of Maisonneuve—is used by most surgeons. The maneuver, which is also termed *divulsion*, is carried out at intervals of from ten to fourteen days; it is a very effective and harmless measure of relief, provided it is carried out with reasonable care and asepsis is observed.

The use of *electrolysis* (Newmann[16]) in conjunction with mechanical dilatation does not seem to possess any advantage over the latter.

Internal urethrotomy is employed in connection with stricture of the straight portion of the urethra when dilatation does not accomplish the purpose, when the stricture is particularly firm in character, or when, for special reasons, haste is desirable. The method is contraindicated in impermeable stricture, urinary infiltration, abscesses

and fistulæ. Strictures located near the meatus are divided with the scalpel; the deeper ones are sectioned with the *urethrotome*. Of the many especially constructed instruments used for the purpose, the model shown in Fig. 1035 is as useful as any. Kollmann[15] uses an

FIG. 1034.— KOLLMANN DILATORS.

5. Kollmann's straight articulated 4-bladed dilator for the anterior part of the urethra, with short branches.
6. " " " " " " " anterior parts of the urethra, with long branches.
7. " curved " " " " " posterior part of the urethra.
8. " " " " " " " " and part of the anterior urethra.
9. " " " " " " " " part of the urethra, with Guyon's curve.
10. " double-curved " " " " " " and part of the anterior urethra, with Guyon's curve.
11. " straight " " " " " anterior part of the urethra, with irrigating attachment.

illuminating urethrotome, which makes the division of the strictured area visible.

The instrument divides the callus in its entire thickness, preferably at the upper aspect of the canal. A retention catheter is left *in situ* for three or four days. It is proper to say that the operation is not largely practiced in this country. Most American surgeons fear the consequences of urinary infiltration and infection — an apprehension the writer is inclined to share.

External urethrotomy (boutonière, sectio perinealis) is employed for the relief of stricture and, as already stated, finds a useful field in connection with rupture of the urethra, the removal of foreign bodies from the canal and of tumors of the bladder.

The measure is indicated in stricture as follows:

(1) Long non-distensible stricture with a rigid callus, especially the traumatic.

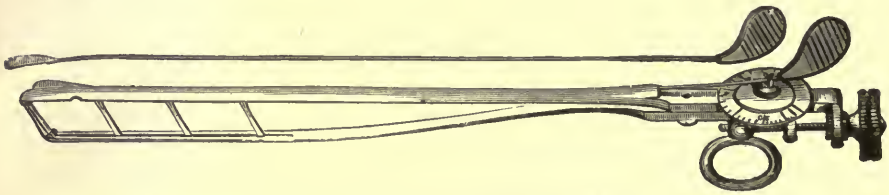

FIG. 1035.— OTIS URETHROTOME.

(2) All strictures where the methods previously stated do not accomplish the purpose.

(3) Strictures with complications, i. e., fistulæ, abscesses, false passages and putrefactive cystitis.

The measure of choice consists in opening the urethra *in front of the stricture,* and division of the latter; in this way the central portion of the urethra is most easily found. The patient is placed in the lithotomy position and the perineum is incised in the exact median line, the incision extending beyond the stricture in both directions, exposing the urethra, into which a guide has previously been inserted. After the urethra is opened it is held apart with tenacula and the strictured part is identified and freely sectioned from before backward. The bladder is drained through the perineal wound.

In not a few cases it is difficult, or impossible, to find the opening in the stricture, especially when the scar tissue is abundant and the urethra is much deformed. If, after a careful search, the urethral

canal cannot be identified, *suprapubic cystotomy and the retrograde introduction of a guide* is permissible.

When a considerable area of urethra is destroyed, reëstablishment of the canal by plastic methods of repair is indicated. In a certain number of cases it will be found necessary to establish a permanent perineal fistula (Mhra,[17] Poncet,[18] Guyon,[19] Delore[20]).

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CHAPTER V

SURGERY OF THE TESTICLE AND SPERMATIC CORD

Undescended and Misplaced Testicle.— There are four types of *undescended testicle:* (1) the testicle in the abdomen at or about its fetal position; (2) the testicle at the internal ring; (3) the testicle in the inguinal canal; (4) the testicle below the internal ring.

A *misplaced testicle* may be situated (a) in the perineum, or (b) on the thigh near the perineal canal. According to Bevan[1] it is feasible to dissect the misplaced testicle from its faulty environment and place it into the scrotum in much the same manner as in the inguinal malposition.

The *dangers and discomfort* coincident to undescended testicle are of sufficient importance to warrant efforts at relief. The subject has been discussed to a considerable extent in connection with hernia (Part XVI, chap. xii). The dangers of the condition are: (1) coexisting hernia; (2) the tendency to serious lesions — epididymitis, atrophy, twisting of the cord and consequent gangrene; (3) injuries from the exposed position of the organ; (4) likelihood of the development of malignant disease; (5) mental disturbances.

While the use of apparatus of various kinds designed to promote post fetal descent of the testicle are employed, none of them seem to exercise a satisfactory influence in this connection.

Of the various methods of *operative relief* that of Bevan[1] seems to meet the indications. Bevan describes the technic as follows: "An incision is made about three inches long over the inguinal canal. This cut should divide skin, superficial fascia, and the aponeurosis of the external oblique. Under the external oblique will be found a pouch of peritoneum extending from the abdominal peritoneum through the canal and down to the scrotum. Sometimes the pouch extends to the bottom of the scrotum, even in cases in which the testicle has never been out of the abdominal cavity. This pouch of peritoneum is covered by the cremasteric muscle and fascia and the transversalis fascia. These thin layers should be divided and the peritoneal pouch opened. The

vaginal processes of peritoneum should be divided transversely well above the testicle. Care should be taken not to injure the cord, and, in children, where the peritoneal process is as delicate as tissue paper, the dissection must be made with great care and with small instruments. After complete transverse division of the vaginal process, the upper end is closed with a catgut ligature, as is the sac in a hernia operation, and the lower end is closed with a purse string suture, making a tunica vaginalis for the testis (Fig. 1036). Then, with a gauze sponge, the peritoneum is carefully wiped off from the cord. As the wiping proceeds the cord will gradually lengthen until the testicle can be brought well down upon the thigh. Tense fibrous strands in the cord are to be torn, either with the fingers or with dissecting forceps, and the cord is to be freed of everything except the vas and vessels. When, as in the exceptional case, a large peritoneal pouch extends to the bottom of the scrotum, the testicle can now be pushed into this and retained by a purse string suture within the neck of the scrotum (Fig. 1037). As a rule, however, it is necessary to make a cavity by blunt dissection with the finger.

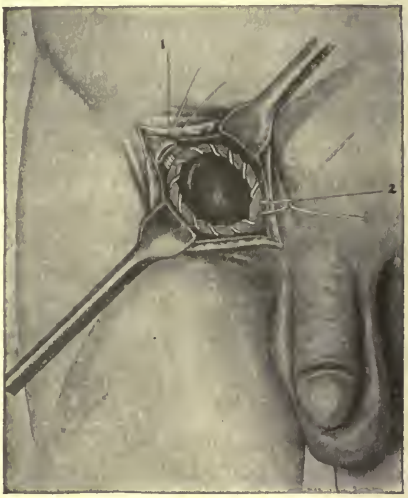

FIG. 1036.— BEVAN'S OPERATION FOR RETAINED TESTIS IN THE INGUINAL REGION.

Shows the vaginal process divided, the upper segment ligated, and the part below with a purse string suture about it ready to be made into a tunica vaginalis (Keen's 'Surgery'').

"In a few cases — but these will be quite the exception — it is found, even after the free exposure of the cord, that it is not long enough to permit of the reduction well into the scrotum. In such cases it will be seen that the shortened structures are the spermatic artery and veins. These can be divided between two ligatures, care being taken to avoid injury to the vas and the vessels of the vas. It will then be found that the testicle can by gentle traction be brought down sufficiently to replace it in the scrotum without tension. The artery of the vas and the veins of the vas are quite sufficient to supply

the entire testicle after the ligation of the spermatic artery and anterior group of veins. The wound is now closed as in ordinary hernia operation, not transplanting the cord, as is done in the Bassini operation, but allowing it to remain in its normal position. The success of the operation depends upon freeing the testicle so completely that it can be placed in a roomy pocket well down in the scrotum without tension."

Among other abnormalities should be mentioned *anorchism* (absence of one or both testicles); rudimentary development, and *supernumerary testicle.* The existence of the latter is doubtful.

Injuries and Diseases of the Scrotum.— Injuries of the scrotum possess certain regional peculiarities. They are likely to be followed by extensive extravasation of blood, filling the scrotum and extending into the perineum and up over the abdomen. While the effusion is usually absorbed, it is occasionally necessary to liberate it by free incision; this is especially true when infection supervenes. If one or both testicles are prolapsed through a wound, they should be replaced and the wound closed, making proper provision for drainage.

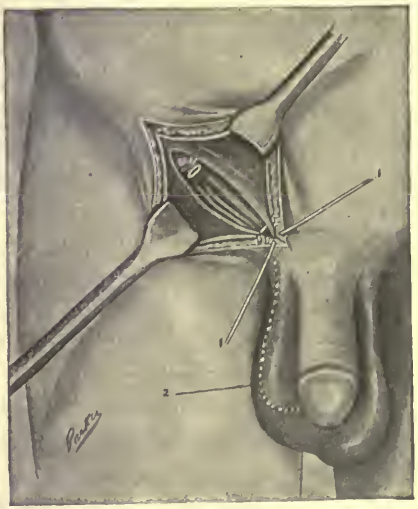

FIG. 1037.— BEVAN'S OPERATION FOR RETAINED TESTIS IN THE INGUINAL REGION.

Shows the testis in scrotum (Keen's "Surgery").

Acute inflammatory diseases of the scrotum include infection of wounds, furuncles, phlegmons, and acute processes following contact with chemical irritants, such as poison ivy, carbolic acid, etc. The integument of the scrotum is particularly susceptible to the action of antiseptics. The various forms of inflammation are treated on general surgical principles.

Elephantiasis is taken up in Part II, chap. x.

Tumors of the scrotum do not differ from those discussed in Part VI. *Melanotic sarcoma* in this region is particularly malignant.

Carcinoma of the skin of the scrotum, aside from the usual forms of the disease, occasionally appears in a special form found in chimney sweeps (p. 1263). It is believed to be due to the irritation caused by soot and is not very malignant. Recently a similar lesion has developed in persons exposed to the X-ray for long periods of time. Early removal of the lesion makes the prognosis favorable.

Injuries and Diseases of the Tunica Vaginalis and Spermatic Cord.—Hematoma may develop within the scrotum after injury to the coverings of the testicle. The effused blood lies between the tunica vaginalis and the infundibuliform, cremasteric and intercolumnar fascia. This is designated as a hematoma. Usually the swelling is pear shaped with the base below. Rest and support of the scrotum is all that is required. If the collection of blood persists or becomes infected, incision and drainage are indicated.

Hematocele is a term applied to an effusion of blood into the tunica vaginalis; this is rare compared to the extravaginal hematoma. It occurs in connection with trauma, but may be pathologic in origin. The *traumatic form* follows contusion of the testicle, but occurs more frequently as the result of hemorrhage following the puncture of a hydrocele or of traumatism of a hydrocele. If the blood clot is not absorbed and the hemorrhage has occurred in a preëxisting hydrocele, the sac is emptied and is then treated as in radical operation for hydrocele.

The *pathological form* of hematocele is due to a chronic hemorrhagic inflammation of the tunica vaginalis with resulting thickening of this membrane. There may be repeated hemorrhages. Traumatic and pathological types of hematocele are not readily differentiated. Hematocele of itself is distinguished from new growth by the aspirator.

The *treatment* consists in the radical removal of the entire mass.

Hematocele of the cord, both traumatic and pathological, occurs in a persistent vaginal process and lies in front of the cord. The lesion is exceedingly rare and differs from similar conditions in the tunica only as to location.

Acute hydrocele testis, as a distinct complication, occurs in the course of an acute gonorrheal epididymitis; and acute inflammation of the tunica, in connection with tuberculous and leutic lesions of the testicle, and in acute infectious diseases, such as mumps, typhoid fever, etc.

The acute onset of the effusion, with swelling, redness, fluctuation,

pain, and its translucency makes the *diagnosis*. In case of doubt, aspiration is permissible.

The *treatment* consists in rest in bed and support of the scrotum by means of a "Bellevue Bridge" (p. 2414) or similar apparatus. The process runs its course in from a few days to two weeks. In cases attended with great tension and pain incision and drainage is indicated.

Suppurative inflammation of the tunica vaginalis may begin as such, or an acute hydrocele may become so.

The *symptoms* are more severe than those of the simple acute hydrocele. Transillumination shows the contents to be only semitranslucent. Persistence of the effusion suggests its purulent character. The constitutional disturbances are those of an acute infectious process, the severity of the symptoms depending upon the character of the underlying cause.

The *treatment* consists in free incision and drainage of the tunica vaginalis.

Acute hydrocele of the cord presents a picture very similar to that of the tunica vaginalis, except that the local manifestations are higher up. The process may resemble strangulated hernia. In doubtful cases immediate exploration is permissible. In clearly defined cases the treatment is similar to that of hydrocele of the tunica testis.

Chronic inflammation of the tunica vaginalis (*hydrocele*) consists in the accumulation of fluid in the tunica vaginalis without acute symptoms. It is a common condition and may occur at any time of life. *Trauma* is the most common cause, the next most frequent one being gonorrhea. Tuberculosis, syphilis, typhoid fever, and neoplasm are occasionally responsible for the effusion and must be taken into account. The aspirated fluid is generally clear and transparent, yellow or amber in color, and from long standing, cholesterin crystals are precipitated. It may be cloudy, in which event spermatozoa may be present in the fluid. The tubercle bacillus has been found in the fluid on many occasions. V. Bramman[2] suggests that the effusion (as in pleurisy) is not infrequently due to tuberculosis of mild degree.

The *symptoms* of hydrocele are insidious in character. The patient first notes either the enlarged scrotum or the discomfort of the drag on the cord. As the effusion accumulates the discomfort becomes serious. The skin is often irritated from friction of the clothing. The tumor is egg shaped and clearly outlined.

The *diagnosis* is made on the history of the case, the presence of

fluctuation, and the translucency of the swelling. It must be differentiated from scrotal hernia. The aspirator clears up the diagnosis.

Spermatoceles usually occur in the form of small cysts near the head of the epididymis. Palpation reveals two distinct masses, the testicle and the spermatocele.

Chronic inflammatory processes of the testicle (orchitis) and epididymis (epididymitis) are not infrequently responsible for hydrocele of small size. If the effusion is large in quantity, it may so mask the underlying condition that the latter is not revealed until the fluid is evacuated.

Hydrocele of the cord is common in children and is due to the accumulation of fluid in a sector of the persistent funicular process. It is often mistaken for hernia. Fluctuation, the blue color, and the translucency of the sac differentiates it from hernia. Aspiration establishes the diagnosis.

Hydrocele communicating with the peritoneal cavity through a patent vaginal process is exceedingly rare. The tumor disappears when the patient assumes the recumbent position. This makes it difficult to differentiate the condition from reducible hernia. The aspirator makes the diagnosis. However, in doubtful cases, exposure of the process is the safer procedure.

In the *treatment*, injections of irritating fluids must be avoided for obvious reasons.

Hydrocele in a hernial sac develops when the neck of the latter is obstructed by chronic inflammation, such as occurs in connection with prolonged pressure of a truss pad.

In *hourglass hydrocele* one sector of the sac is extra-, the other intra-abdominal. The communicating neck lies in the inguinal canal. A hydrocele of this sort may attain gigantic proportions (Bevan[1]).

The *treatment of hydrocele* is divided into (1) palliative and (2) radical; (2) may be divided into (a) injection treatment and (b) operative treatment.

The *palliative treatment* consists in withdrawal of the fluid with the trocar and cannula, repeating as often as necessary. The measure has a limited field of usefulness.

The *injection treatment* is effective in many instances. The older iodin method has been displaced by the injection of 95 per cent carbolic acid. The technic is as follows: The scrotum is sterilized by shaving and scrubbing with soap and water and alcohol as for any aseptic operation. A half dozen drops of one per cent solution of

cocain are injected into the scrotal integument in front and a little above the center of the swelling; through this cocainized area a fairly large trocar and cannula are pushed into the cavity of the tunica vaginalis. The position of the testicle, which is posterior, should be kept in mind and care must be exercised not to injure this organ with the trocar (Fig. 1038). The trocar is withdrawn and the cannula is pushed well to the upper limit of the sac and the fluid is allowed to escape. It is important that all the fluid be withdrawn, and in order to insure this it is best to withdraw the last of it with a half ounce aspirating syringe, which fills the cannula. Leaving the cannula in place at the upper part of the sac, from five to twenty drops of a 95 per cent solution of pure carbolic acid are now injected into the sac and the cannula is withdrawn. As this is done the part of the sac through which the cannula is passed should be firmly grasped with the thumb and finger and held for four or five minutes, while with the other hand the sac is kneaded, so as to bring the carbolic acid in contact with all parts of the wall of the hydrocele. Following the injection, there is usually a very brief period during which there is pain and burning, but this soon subsides.

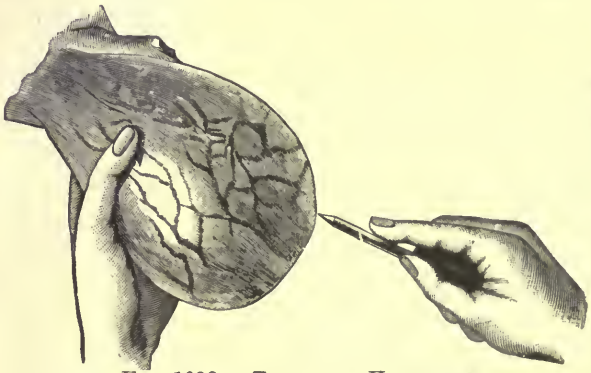

FIG. 1038.— TAPPING A HYDROCELE.

The patient should be advised to remain quiet and at home for a few days. A certain amount of reaction follows the injection. Usually the scrotum swells up to about half the size it was before the withdrawal of the fluid. This swelling gradually disappears, and in successful cases the fluid which collected during the reaction is entirely absorbed in from three to six weeks. The treatment, if properly employed, is successful and the lesion is cured by one injection in more than eighty per cent of the cases. A second or even a third injection may be necessary, and may be successful. The injection treatment is indicated only for those cases which have a thin walled sac;

in the long standing cases with thick, indurated sacs it usually fails.

The *operative treatment* most largely practiced today is the so-called "bottle operation" of E. Wyllys Andrews,[3] who describes the procedure as follows: "An anterior scrotal incision is made as in the older methods (through the scrotum in its long axis). The skin should be held tense and the dissection should be nice to the exact layer which will enucleate the translucent bladder-like mass from its bed.

"Careful study of the funicular part of the sac is now to be made. Usually a little funnel continues one or two cm. up the cord. The

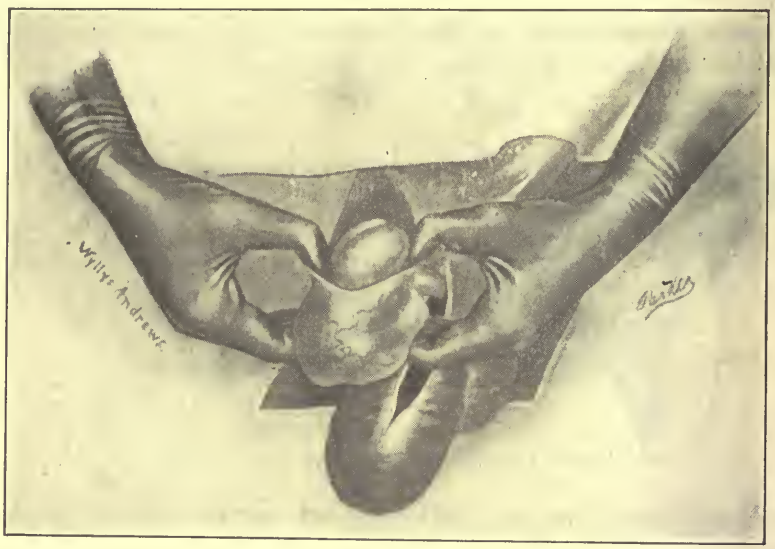

FIG. 1039.—ANDREW'S "BOTTLE" OPERATION (Keen's "Surgery").

extreme upper end of this marks the beginning of the cut made into the sac. This cut is vertical on the anterior border and only about two cm. long. We enlarge it a little by stretching. Sometimes it is wholly confined to the part over the cord. The incision in the sac is prolonged to its extreme upper end along the cord, if the first cut did not do this. When the sac has been emptied, it is like a bottle or bag, with a small hole at the top. Dilating this slightly with one or two fingers, the orifice is held open and the testis is pushed up into it with the other hand or the two thumbs (Fig. 1039). In a moment it can be squeezed through, and the whole sac will be instantly everted

with the small buttonhole so closely surrounding the cord that it is scarcely visible. The short incision contracts, so as to fit around the cord, and the whole sac by its elasticity seems to collapse around the epididymis with its white serous surface almost as smooth as that of the testis. I have never seen the everted sac voluminous enough to lie in wrinkles or folds, but no harm would result if it were so to act. Ordinarily, the largest hydrocele sacs when collapsed assume about the size of the testis, showing that their walls remain perfectly elastic.

"The skin is quickly closed with clips or light suture, without drainage. Such wounds heal within a week. The patient gets about on the third or fourth day, sometimes earlier. The amount of swelling about the testis is usually small, even in double hydroceles. Tenderness and pain are moderate or absent, and no fever and malaise are felt after the second day."

Simple hydrocele of the cord is treated by the injection method. When a communication with the abdominal cavity exists, the sac is best excised.

In the *hourglass* type, Bevan[1] incises the sac in the scrotum, stitches it to the skin, and introduces a drainage tube to the extreme limits of the upper sac. The tube is gradually shortened as the sac contracts, and becomes obliterated by adhesive inflammation set up by the tube. The sacs are irrigated daily with a sherry colored iodin solution, which insures asepsis and at the same time provokes the necessary degree of obliterative inflammation.

Varicocele (Fig. 1040) consists of a dilatation and elongation of the veins of the spermatic cord, associated at times with *atrophy of the testicle;* it is a common condition. The spermatic veins (pampiniform plexus) are chiefly involved. If unilateral, the left testicle is usually involved. The most plausible explanation of this fact is the anatomic difference in the right and left spermatic veins, the right emptying obliquely into the vena cava, and the left at right angles to the renal veins (Curling[4]).

The *diagnosis of varicocele* is usually self evident. As a rule, the beginning of the condition is not attended with symptoms. As the veins dilate the scrotum sweats inordinately and a burning pain develops; this radiates into the groin and is increased by prolonged standing. The pain is usually neuralgic in character. Lessening of the sexual appetite is probably imaginary. Schwartz[5] seems to have shown that varicocele does not cause disturbances except when an inguinal hernia is present; this view is shared by a number of surgeons,

especially those who examined a large number of recruits during the recent "world war."

Examination reveals an enlargement of the scrotum (usually on the left side) which is greatest at its lower aspect. The dilated veins are frequently visible through the thinned out skin. The testicle is small and the veins feel like a "bag of angle worms." When the patient

FIG. 1040.— VARICOCELE (Johnson).

assumes the recumbent position the mass disappears; it reforms *from the bottom* when he stands erect.

The *treatment* of varicocele must take into account that *spontaneous recession* of the process ocasionally occurs, especially when a suspensory is worn. Vince[6] amputates the redundant scrotum with the view of obviating the drag on the cord; the method is not widely practiced.

Ligature and resection of the dilated veins is indicated in advanced

cases. In view of the causative influence of an enlarged inguinal canal, the following procedure is regarded as best suited to the purpose (Narath[7]). The internal spermatic vein is exposed in the inguinal canal, doubly deligated and cut. The canal is closed as in Bassini's herniotomy. Following this the paminiform plexus is exposed and treated as follows:

The vas and its vessels, both veins and artery, are separated from the spermatic vessels. About two inches of the spermatic vessels are now separated from the vas and its vessels by blunt dissection. If the spermatic artery can be recognized, it should be avoided and not included in the ligature. Very frequently it is included, and apparently without doing any damage to the testis or interfering with its function. About two inches of the veins are included between two catgut ligatures. The portion between the ligatures is now resected and the ends of the ligatures are tied, shortening the cord somewhat.

Injuries and Dislocation and Torsion of the Testis.— *Injuries* of the testicle are very painful and usually are attended with nausea, vomiting, and the symptoms of shock; the last is occasionally fatal. More or less extravasation of blood and swelling usually occurs.

The testicle may be *dislocated* by blunt force into the inguinal region, on the anterior surface of the thigh, or into the perineum. If reduction is not effected the organ may become fixed in its new position by adhesive chronic inflammation; in this event it must be dissected from the embedding fibrous tissue before reduction can be accomplished.

Abnormal movability of the testicle is not a common condition (in an anatomical sense); the organ can be pushed up on the groin and even into the inguinal canal. The condition is due to the existence of a large pouch of peritoneum, which fills the scrotum, and in this the testicle is suspended; it predisposes to torsion of the cord.

Torsion of the testicle occurs suddenly after injury or after a violent muscular exertion, and presents a clinical picture not unlike that of strangulated hernia. Operative exposure makes the diagnosis. If the torsion cannot be corrected, castration becomes necessary.

Acute Inflammation of the Testis and Epididymis.— In addition to traumatic orchitis and epididymitis these parts are often visited by infective organisms. Two types are differentiated, those of urethral and those of metastatic origin.

The *urethral form* is by far the most common and is due to extension of an acute inflammatory process along the vas to the epididymis

or testis. In most instances the infection is gonorrheal, although the pyogenic bacteria are also responsible for the condition; in the latter instance instrumentation plays an important rôle.

The *metastatic form* occurs in connection with acute infectious diseases, especially mumps and typhoid fever. In this form the testicle is usually primarily invaded, and the epididymis is involved by extension.

The *clinical picture* is that of an acute inflammation; the scrotum is often edematous. The duration of acute symptoms lasts for a week or two. The symptoms gradually disappear, generally leaving, however, for some time, a hard nodule in the head or tail of the epididymis; this may persist, and if it does the epididymis is permanently blocked. If the infection is bilateral the patient is left sterile.

FIG. 1041.— ADHESIVE PLASTER BRIDGE, A SUPPORT FOR THE TESTIS IN EPIDIDYMITIS (Bellevue Bridge).

The *treatment* consists in rest in bed, with proper support of the testicle by means of the "Bellevue Bridge" (Fig. 1041) or a triangular bandage. Hot fomentations or icebags are used, if they add to the comfort of the patient. After the acute stage a properly fitted suspensory should be worn until all signs of the disease have disappeared. In a few cases suppuration develops, and incision and drainage become necessary.

Chronic Inflammation of the Testicle and Epididymis.— Tuberculosis, next to gonorrhea, is the most common disease of these organs. In the great majority of cases it develops in the epididymis and later extends to the testis (Fig. 1042). The tubercle bacilli enter the epididymis through the circulation from a primary focus elsewhere in the body. Tuberculosis in this situation rarely occurs by direct extension. Local trauma and previous infection favors the lodgment of the tubercle

bacilli in the epididymis. The disease begins as a subacute — rarely as an acute — process. A mixed infection often occurs in the later cases. A hard lump develops in the head or tail of the organ and within a few weeks extends along the vas, producing a nodular thickening of the latter. The tunica vaginalis is often involved, becoming more or lesss distended with fluid. As a general rule, in a few weeks or months, the process extends to the testis proper and to the surrounding structures of the scrotum. The scrotum finally becomes attached to the testicle and fistulae are formed, through which caseous material from the focus in the epididymis is discharged. Extensive involvement may produce a projection of a fungous mass of tuberculous granulation tissue. The disease usually extends along the vas to the seminal vesicles, prostate, bladder, and to the other testicle.

FIG. 1042.— TUBERCULOUS TESTIS AND EPIDIDYMIS.

Note thickening in the epididymis.

The *diagnosis* usually is not difficult. The subacute onset, the physical examination, and the finding of fistulae is sufficient evidence.

The *treatment* of tuberculosis of the testicle is essentially hygienic. Operative efforts at relief are unsatisfactory, unless the disease is distinctly limited to the testicle, when castration is indicated.

Lues of the testicle may be congenital. In the adult it is essentially a tertiary manifestation. It is usually located primarily in the testicle itself in the form of a gumma, in contradistinction to tuberculosis, which usually begins in the epididymis. Occasionally the disease appears in the form of an infiltration.

Lues of the testicle runs a painless course; the organ gradually increases in size. If the process attains large dimensions, the drag on the cord causes much discomfort.

The *diagnosis* must per force be based on the blood test and thera-

peutic test to differentiate the process from tumors and tuberculosis.

Actinomycosis and leprosy seldom involve the testicle.

Spermatocele and Retention Cysts.— Spermatoceles are cysts containing spermatozoa. They are situated near the head of the epididymis, and often are placed between it and the testis. They may attain the size of an egg. The cyst originates in a fetal remnant in the region of the testicle and epididymis.

A *retention cyst* may also develop from these fetal remnants, and occasionally contain spermatozoa. The position of the cysts and their slow, painless development make the *diagnosis*. Unless they attain considerable size, they do not cause disturbance. Possible etiological factors are trauma and inflammation. Calcareous, or even bony, infiltration of the cyst wall may occur. Aspiration and injection of iodin may be followed by obliteration of the sac; failure in this justifies the radical operation.

Tumors of the Testicle.— Almost all varieties of tumors may occur in the testicle, but are not common. Lipomata, fibromata, enchondromata, and osteomata are rarely encountered.

Lipomata in the scrotum occur, but as a rule are located in the cord.

Sarcomata are fairly common and occur in children and early adult life. They may commence either in the testicle or the epididymis and usually appear as small, round cell, or spindle cell, sarcoma. Melanotic sarcomata are very rare. The course of the disease is usually rapid; it extends along the spermatic cord to the abdominal cavity. At times a sarcoma ulcerates through the scrotal tissue and presents a large fungus mass. The prognosis is not good, even after early radical removal.

Carcinomata are far more common than sarcomata. They may be either the medullary or the scirrhous type. Early invasion of the inguinal and the retroperitoneal glands occurs. The *diagnosis* is made by the hardness and irregularity of the growth, and the absence of acute symptoms. In case of doubt, operative exploration is permissible.

The *treatment* consists in castration and the removal of the glands.

The *prognosis* is not good.

Dermoid cysts and teratomata of the testicle are not rare. They are not recognized until exposed by incision. Tumors of this sort are usually benign in character, but occasionally they become malignant.

The *treatment* consists in removal of the growth without sacrifice

of the testicle; this is often feasible when the effort is made early in the process

Castration is often required in connection with various lesions of the testicle. The operation is performed as follows: An incision is made beginning over the external ring and extending down the scrotum for about three inches. This divides the skin and dartos. In cases where the skin is involved an elliptic incision, including the diseased part, is made. The testicle and cord, with their coverings, are separated from the scrotal tissue. If the condition demanding castration is one in which the cord may be involved, as in tuberculosis or malignant disease, the inguinal canal should be split up and the cord ligated as high as is possible before division. The ligation of the cord must be done thoroughly. It is well to transfix the cord at an avascular part and double ligate and even put on a second ligature around the entire cord for additional safety. In the aseptic cases the wound is completely closed, with the exception of a small drain of gutta percha, which is removed at the end of twenty-four hours. In the infected cases drainage must be provided.

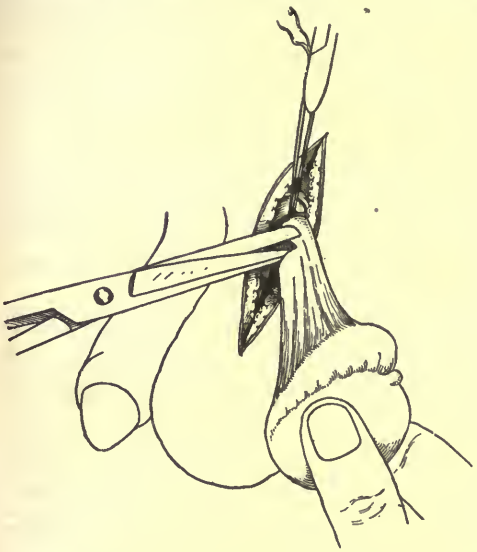

FIG. 1043.— ORCHIDECTOMY.

Shows a temporary ligature about the cord and the scissors cutting through below it. The arteries are then ligated individually and the temporary ligature severed.

Diseases of the Seminal Vesicles.— Inflammation of the seminal vesicles (vesiculitis) of an *acute* character is usually a gonorrheal or mixed infection by extension from the urethra. It is not always feasible to differentiate mild spermatocystitis from prostatitis. The enlarged vesicles may occasionally be felt through the rectum. Pain in the perineum is likely to be severe.

Chronic spermatocystitis may evolve from an acute attack, but is likely to be chronic in its onset. The vesicles are enlarged and may become *dilated* or even *cystic;* in neither case need the ejaculatory ducts be occluded. The vesicles are usually filled with a dark, brown

fluid, and may contain concretions (phosphate of lime). In protracted cases the vesicles gradually atrophy or, when the process is suppurative in character, fistulous communications with the rectum may be established.

Vesiculitis is usually attended with inflammation of the prostate and urethra. This may be responsible for urinary obstruction and is not infrequently attended with painful spasm suggesting renal colic (false renal colic of Picker). In a small number of cases a pyogenous process is responsible for peritonitis, periproctitis, and ischiorectal abscess. Völcker[8] has attempted to show a causative relationship between gonorrheal vesiculitis and rheumatism (gonorrheal). Fuller[9] agrees with this view, but believes that pyogenic infection may also be responsible for the joint lesions.

The *clinical manifestations* of spermatocystitis are not characteristic of the disease. The symptoms consist in indefinite pain in the perineum radiating in all directions. Völcker[8] regards spasmodic pain along the vas as diagnostic. A urethral discharge may be absent. The import of sexual disturbances is doubtful.

The *diagnosis* cannot be based on the symptoms. It must be established by microscopical examination of the semen, palpation of the vesicles (per rectum), and a carefully conducted urethroscopic examination. Aspiration is permissible in doubtful cases.

The *treatment* in acute vesiculitis is one of non-interference. The patient is kept in bed. Threatening general infection justifies drainage of the vesicles. Massage in the acute stage of the disease is contraindicated.

On the other hand, in the chronic form, evacuation of vesicles by massage possesses considerable therapeutic value. The high location of the vesicles makes the maneuver difficult, unless suitable apparatus is used for the purpose (Morton[10]). In obstinate cases operative measures of relief are justified.

Tuberculosis of the vesiculae seminales is a frequent complication of a similar process in the epididymis; it is rarely primary.

The infected vesicle may enlarge to the size of a walnut. The process thickens the wall of the organ and appears as a mixture of infiltration and caseation. Liquefaction is unusual.

The *diagnosis* rests upon the presence of tuberculosis in the epididymis and vas.

The *treatment* consists in removal of the infected testicle and vas. This is often followed by recession of the process in the vesicle. Di-

rect surgical attack upon the vesicle is not followed by healing (v. Bramman-Rammstedt[2]).

Operations Upon the Seminal Vesicles.— Aspiration of the seminal vesicles is performed through the rectum or perineum. The *rectal route* presents no difficulties; the rectum is opened by means of a speculum and the needle plunged into the palpable vesicle. In the *perineal approach* the needle is entered three centimeters from the median line

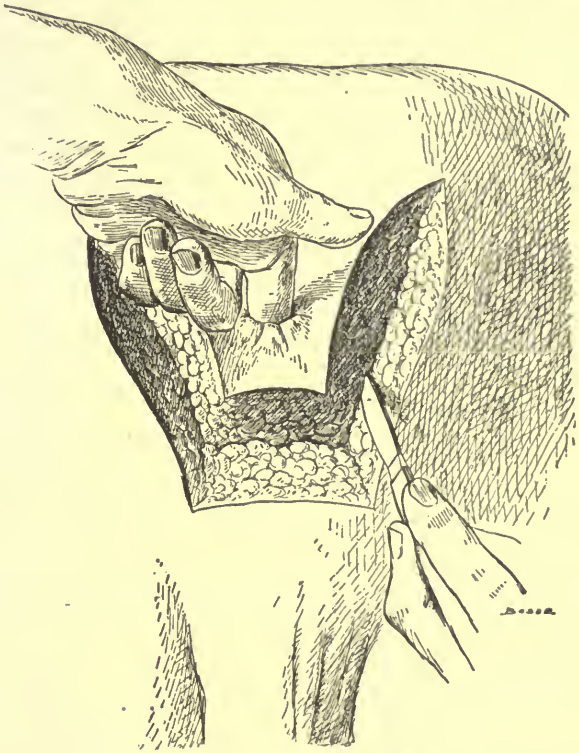

Fig. 1044.— Vesiculotomy.

Showing the manner of making the incision (Fuller's operation).

at a point equally distant from the anus. The finger in the rectum guides the needle to its goal. If pus flows from the needle the knife may be carried along the latter and the vesicle drained.

Incision of the vesicles (vesiculotomy) is advised by Fuller.[9] Morton[10] describes the procedure as follows: "With the patient in the knee chest position, two longitudinal cuts through the skin and fascia are made, opening up the ischiorectal fossae. These are joined by a

transverse incision just in front of the anus. With the finger in the rectum as a guide, the rectal wall is separated from the prostate and vesicles by blunt dissection with the finger tip of the other hand. After the separation is made a long, grooved director is thrust through the tissues into the apex of the vesicle, using the finger in the rectum as a guide. A scalpel is passed along the grooved director and the vesicles laid open for half an inch; the incision in the vesicle is then divulsed, with the finger tip opening up widely the sac.

"The opposite vesicle is opened in the same way. If granulations are found in the cavities they are curetted and packed with gauze and drainage is provided for." Healing by granulation occurs in about three weeks. Squier[11] drains the vesicle through the perineum. Belfield[12] opens the vas in the scrotum and drains the vesicles in this way.

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